RADIOLOGY

A MONTHLY JOURNAL DEVOTED TO CLINICAL RADIOLOGY AND ALLIED SCIENCES
PUBLISHED BY THE RADIOLOGICAL SOCIETY OF NORTH AMERICA

VOL. 28 MARCH, 1937 NO. 3

ERRORS IN X-RAY DIAGNOSIS OF INDUSTRIAL INJURIES

By W. WARNER WATKINS, M.D., F.A.C.R., Pathological Laboratory, *Phoenix, Arizona*

THIS paper and the exhibit accompanying it are not presented with the idea of bringing anything new by way of technic or interpretation to the roentgenologist. That would be impertinent, because the illustrations come out of the very kindergarten of diagnostic roentgenology. Commonplace as they are, however, these errors can be useful if they help us to sense more keenly some dangers which beset our special field of work, and make us more determined to insist on fundamental principles in the practice of x-ray diagnosis.

If, by imagination, we can picture diagnostic roentgenology as an organism, subject to forces which can help it or injure it, as all organisms are, let us consider certain elements of a malignant chronic disease which afflicts us. This ailment is not new and has been treated by many palliatives, but like other insidious malignant conditions which concern us as radiotherapists, whenever we succeed in stopping its advance in one direction, it crops up in a new form and place. We will call this disease "*pseudo-roentgenology*" and will briefly consider its causes, symptoms, and treatment.

There are three causes which spring from a common stalk of normal development; that is, the laudable desire to make the greatest possible use of the most valuable single aid we have in the diagnosis and treatment of human ailments, namely, x-ray. From this normal stalk have sprouted three lines of activity which have become parasitic and now threaten the very life of the organism on which they have grown.

1. The widespread distribution, by energetic manufacturers, of small and inexpensive x-ray plants. In the development of small x-ray units, the slogan of the manufacturers might well be "an x-ray machine in every doctor's office," and is being enlarged to include the office of every irregular practitioner who can legally operate one. A doctor can hardly get his sign on the door, in any hamlet in the United States, and his name in a telephone directory, before some high pressure salesman is importuning him to boost his practice by doing his own x-ray work. "Why pay the radiologist on the next floor or in the next block to make your x-ray pictures? Your office girl, in forty-eight hours, can learn to make perfectly satisfactory x-ray films which you can easily interpret yourself; you can then do your own industrial x-ray work, set fractures under the fluoroscope, examine the hearts, chests, and stomachs of your patients, make a great impression and pay for your equipment inside of a year." Is that an overdrawn statement of what occurs? If it has not been done repeatedly in your own community, you have been overlooked and have, so far, been fortunate. Six definite instances could be cited in which statements even more presumptuous than this

[1] Presented before the Radiological Society of North America at the Twenty-second Annual Meeting, at Cincinnati, Nov. 30–Dec. 4, 1936.

have prefaced the sales of x-ray equipment in a community of fewer than 100,000 people, each of these to be operated by general practitioners who were to make their own interpretations. A most astounding instance of high pressure salesmanship was that in which our Industrial Commission was persuaded to buy an x-ray plant and install it in the office of their medical referee without consulting him and while he was in New York City doing postgraduate work. His successor gave the equipment away, as he needed the room to hold the filing cases of mistakes made by other owners of x-ray equipment.

2. Out of this mistaken idea, created by advertising and propaganda, has developed the next causative factor of the malignant disease of pseudo-roentgenology. Having acquired the equipment, the doctor naturally tries to handle as much of his x-ray work as possible, thinking, in his ingenuousness, that the taking of films of bone injuries and the interpretation of findings is a very simple matter. So, we find many doctors, entirely unfamiliar with the normal appearances of bones and joints on roentgenograms, x-raying their own patients, trying to interpret the films, and basing their reports and management of injuries on these inadequate examinations and frequently incorrect interpretations.

3. The third cause of the disease of pseudo-roentgenology is akin to the other two. It is the still prevalent misconception of the true place and scope of diagnostic roentgenology in the practice of medicine in general. This misconception is well illustrated by a recent decision of the Supreme Court of California which is, in effect, that the practice of diagnosis by x-ray can be indulged in by anyone and does not constitute practice of medicine. The court's decision referred to a chiropractor and stated that he can *not* give colonic irrigations nor diathermy treatments, because these are a part of the practice of medicine, but that he can make x-ray films and interpret them in diagnosis.

If educated jurists have no better conception of roentgenology in relation to medicine, is it any wonder that the average doctor, notoriously credulous to any sales talk, is easily persuaded that his opinion is as good as that of any roentgenologist on what is shown by x-ray films? The courts accept his opinions and interpretations, so why should he harken to the counsel of the jealous x-ray specialist (who doubtless is only trying to save the work for himself), that roentgenologic interpretation requires expert knowledge, a constantly changing technic, and a profound knowledge of clinical medicine?

What are the symptoms or evidences that such a disease flourishes in the organism of roentgenologic practice? While it pervades every branch of our work, this discussion will be confined to some evidences found in a single narrow field, namely, that of industrial injuries, including automobile accidents covered by personal liability insurance. The symptoms arising out of the causes stated above are represented by errors in diagnosis, due (1) to inadequate and imperfect technic, and (2) to interpretations based on insufficient knowledge and training.

The knowledge of skeletal anatomy acquired in medical schools is not sufficient to become the sole basis of roentgenologic interpretations of injuries to bones and joints. Examples of errors due to inadequate technic are familiar to every roentgenologist, but through a unique system adopted by the medical referee of the Arizona Industrial Commission, an opportunity has been given to see an unusual number of them within the past few years. In spite of strenuous protests from many of the doctors involved, the rule was adopted that all reports on industrial injuries treated throughout the State must be accompanied by any x-ray films taken, whether these were regarded as showing injury or not. This has resulted in the accumulation of a large number of mistaken diagnoses including injuries actually shown by the films taken but not recognized by the surgeons who took them, as well as misinterpretations of shadows. In addition to this accumulation of films which

now fills the room formerly occupied by the white elephant x-ray plant, there is another unique set-up in Arizona. This is the Medical Advisory Council to the Industrial Commission, composed of five doctors appointed by the president of the State medical association, to which group was added a consulting radiologist who is a diplomate of the American Board of Radiology. This Council is entirely unbiased, is placed under no restrictions by the Industrial Commission, being asked to examine the injured workmen referred to them as purely medical or surgical problems. The question of the compensation involved in their decisions is not to be considered by them, unless it has a bearing on the physical condition found. If the Council asks for further time for the injury to become stationary, if they suggest medical or surgical procedures not yet tried, if they desire special examinations which no member of the Council can make, these recommendations are followed by the Commission without question, regardless of cost. If the injury is stationary, a rating of permanent disability is given and this is accepted by the Commission, regardless of the shock to the compensation fund when the rating is high, or the frothing and fuming of the workman and his attorney, when the rating is lower than their demands. Of course, the acceptance of the conclusions of this Council as final, in preference to any amount of opinion testimony, pro or con, from experts hired by one side or the other, has not passed unchallenged by the legal ambulance chasers and a concerted effort is being made to secure the abolishment of this Council. It was even made an issue in our last gubernatorial campaign, and since the candidate for governor who publicly attacked the Council's work was elected, the system may not last much longer. A test case has already gone up to our Supreme Court and the system was not only upheld by their unanimous decision, but was highly commended as the only procedure in vogue which is calculated to arrive at a fair and unbiased conclusion.

The errors found as the result of inadequate technic and inexpert interpretation may be grouped under four heads:

(*a*) The cases in which perfectly normal structures have been mistaken for abnormalities by surgeons unfamiliar with x-ray appearances. It would probably seem that fellows of the College of Surgeons ought to be able to interpret films of the extremity bones, at least. However, we have seen more errors in the interpretation of conditions in the wrists and feet than we have anywhere else in the body. A case which caused great commotion in our Industrial Commission had reference to a sesamoid bone of the great toe. The foot was x-rayed and diagnosed by a surgeon as showing a "dislocated (*sic*) sesamoid bone" of the great toe! The case cost the Commission $2,500, and when the films were examined by a roentgenologist, he found the bones, including the sesamoids, entirely normal.

In another case the normal sesamoid in the popliteal space was diagnosed as a fragment of bone *inside* the knee joint. Only the insistence by the consulting roentgenologist that the structure was perfectly normal and entirely outside of the joint, prevented an attempt to remove this by surgical procedure.

One who does not recognize the osperoneum as a normal bone would certainly be classed as a tyro, yet we have seen one reported as a fracture of the cuboid. The mere fact that it was large and irregular in shape did not excuse this error in any one pretending to make x-ray interpretations.

Perfectly normal ununited transverse processes of the first lumbar vertebra (Fig. 1) have several times been diagnosed as fractures by industrial surgeons in our experience. The statement in Gray's Anatomy, that this "anomaly is rarely met with" is misleading. During the past three years we have kept watch of all our films showing this area of the spine, and have found the anomaly present in about 5 per cent of adult spines. Two cases of this sort have gone to court with reputable

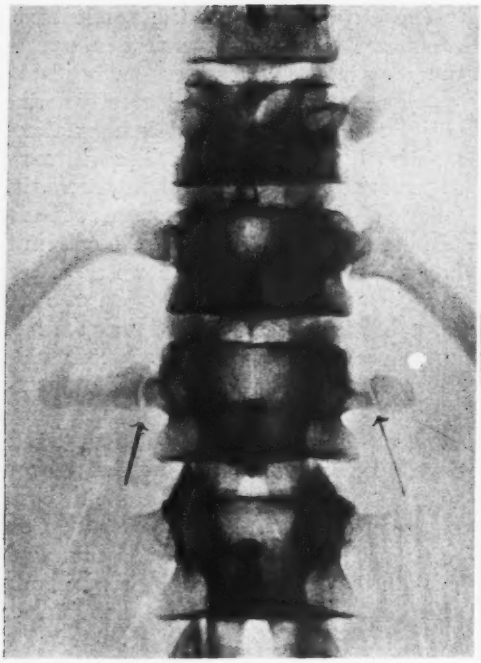


Fig. 1.

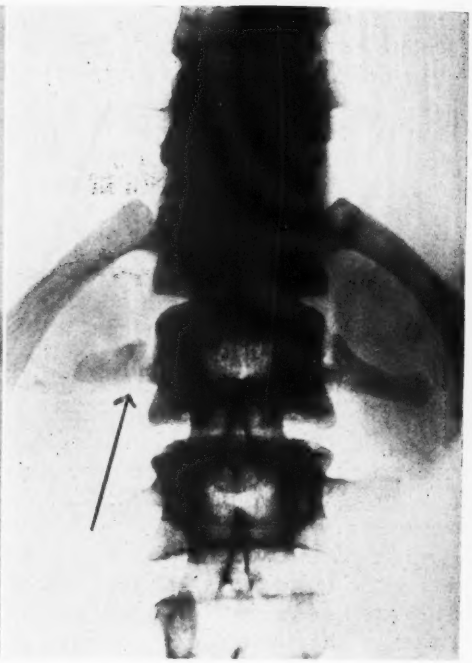


Fig. 2.

Fig. 1. Ununited transverse processes of the first lumbar vertebra, bilateral. Incidental finding with no history of injury. This condition is found on one side or both in approximately one person in twenty.

Fig. 2. Unilateral ununited transverse process of first lumbar vertebra, found after a back injury. Case finally went into court and a surgeon with little experience in x-ray interpretation testified that, in his opinion, this was a fracture. Although an experienced roentgenologist and two competent orthopedic surgeons testified that it was a normal condition for this individual, a verdict for $6,000 damages was awarded. This kind of injustice results from our archaic legal system which permits an uninformed jury to choose the testimony they will accept, regardless of the comparative training and experience of the doctors testifying.

and experienced surgeons testifying that they were fractures (Fig. 2).

The os trigonum has been repeatedly mistaken for fracture by surgeons trying to function as x-ray experts (Fig. 3). Only the roentgenologist whose eye has become thoroughly trained in an appreciation of bone contour, texture, and density can be trusted to say whether such a detached bone is really an os trigonum or a fractured posterior process of the talus. Repeated experience builds up in the brain of the roentgenologist the ability to become certain through steps which he cannot demonstrate to juries nor even to other medical men whose perceptive abilities are not similarly trained. In the case of the os trigonum, as with other conditions, it is often just as difficult for a roentgenologist to show *why* he is certain of his opinion, as it is for the expert in precious stones to demonstrate to the untrained eye why he knows a diamond is real and not an excellent counterfeit. We may listen solemnly and nod our heads but, in the end, we must simply take his word that it is so.

The tibiale externum has been a source of error several times in our experience and in at least one instance such a bone has been removed surgically as an ununited fracture, without benefit to the patient.

The free ulnar styloid has several times been mistaken for fracture. When associated with Colles' fracture this error is not important, but when such a bone is the only basis for diagnosis of injury, it becomes serious.

Free bones about the humeral condyles

or beyond the malleoli at the ankle have been mistakenly diagnosed as fractures several times.

Fig. 3. An os trigonum which was diagnosed as fracture by an orthopedic surgeon who makes his own x-ray interpretations. The case finally had to be adjudicated by the Medical Advisory Board, who decided it was not a fracture.

The anomalies found so frequently in the lumbosacral area will be mentioned only to insist that the significance of the conditions found belongs in the realm of roentgenologic interpretation, and the roentgenologist should not yield to the orthopedic surgeon the entire right to say whether or not certain anomalies found are likely to be the cause of the symptoms complained of. The roentgenologist is primarily a clinician and has built his special knowledge on general medicine. In drawing his conclusions, he should use all the clinical knowledge he possesses, as well as his technical skill and experience as an expert in interpretation of x-ray shadows.

A familiar anomaly illustrates what might happen in the automobile liability racket. A man was driving with a friend and their car collided with a truck. The friend claimed a back injury and sued the company owning the truck for $50,000.

Fig. 4. An unfused tip of the spinous process of the fifth lumbar vertebra, associated with spina bifida occulta of the sacral arch. This condition was found during x-ray examination after an automobile accident and was diagnosed as fracture by a surgeon. This case finally went to court, suing for heavy damages. The jury accepted the unsupported testimony of a roentgenologist that the condition was an anomaly, as against the contentions of three surgeons that it was a fracture and brought in a verdict of "no damages."

The chief evidence of injury, aside from his complaints, was a film showing a condition diagnosed as fracture of the spinous process of the fifth lumbar vertebra (Fig. 4), this being the testimony of the man who made the film and three surgeons. The condition shown was not a fracture, but a congenital anomaly associated with spina bifida.

Belonging in this same group are the errors of misinterpreting epiphyseal lines as fractures. This still happens, in spite of all the voluminous literature on the subject, this evidently not being as widely read as it should be. The normal epiphyses which later are going to unite, differ from the anomalous bones mentioned above which remain permanently ununited. In this group, we have seen instances of the following:

The internal humeral epiphysis, which is frequently seen in young workmen (Fig. 5).

The tubercle of the fifth metatarsal bone.

Prominent tibial tubercles, diagnosed as Osgood-Schlatter's disease.

The marginal rim of the ilium, which may develop in several pieces on one side and in a solid piece on the other.

The upper margin of the acetabulum, which may remain unattached as the os acetabuli.

The extremity of the acromion process.

Other transverse processes in the spine beside the first lumbar.

Tips of articular processes in the lumbar spine.

(*b*) The second group of errors due to inadequate technic are those in which the examination was too limited in scope, or in which inexperience has caused the lesion actually shown to be overlooked. This group will illustrate the danger of trying to separate technical procedures from professional services in hospitals. In practically every case coming before our Medical Advisory Council, the roentgenologist has called for some additional examination or some special technic not previously employed, and some astounding revelations have been uncovered. Perhaps the most striking illustration of the necessity of combining interpretation with special technical procedures directed by the roentgenologist and basing both of these on a knowledge of the clinical symptoms, was the following case: A policeman got up suddenly out of a swivel chair and stepped on a match which rolled under his foot. He fell, striking his elbow on a concrete floor and bruising his chest on the edge of a large badge which he wore just below his left clavicle. X-rays of elbow and shoulder were made, and a crack in the head of the radius found. This healed, but symptoms of pain and muscular weakness persisted, which were diagnosed as brachial neuritis. His case finally came to the Advisory Council. A review of the films was made by the roentgenologist, who found a fracture of the first

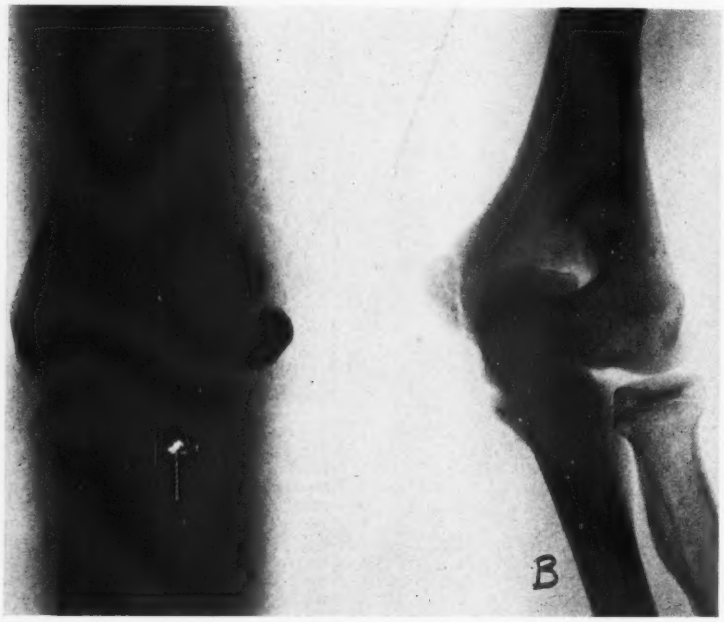


Fig. 5. In (*A*) there is a true fracture of the internal condyle of the humerus with the usual displacement distalward. In (*B*) fracture of the internal condyle was diagnosed, but this opinion was changed by the surgeon when he consulted a roentgenologist, who demonstrated under the fluoroscope that the fragment was not movable and not tender over the line of separation.

rib about one inch from the sternal end, the larger fragment being displaced downward (Fig. 6). Since the symptoms suggested involvement of the cervical plexus, a survey of the cervical spine was made by x-ray. On one of the oblique views a small cervical rib was found (Fig. 7), which cannot be seen on any other view of this spine or thorax. The final conclusion was that the non-union and depression of the fractured first rib had resulted in pinching the cervical plexus between the two structures, and the relief of this by the proper surgical procedure was advised.

Fig. 6. Fracture of first rib near its sternal attachment, with displacement downward of bony portion. This film was made at the time of the accident but this fracture was not discovered until the case was reviewed by a consulting roentgenologist several months later. More comprehensive examination revealed the cervical rib shown in Figure 7.

In another case of suspected injury to the twelfth dorsal vertebra, three x-ray examinations had failed to disclose positive evidence of injury. Finally, the consulting roentgenologist requested oblique views through the lower dorsal area. One of these showed a fractured transverse process of the last dorsal vertebra—evidence that a serious injury had certainly been sustained.

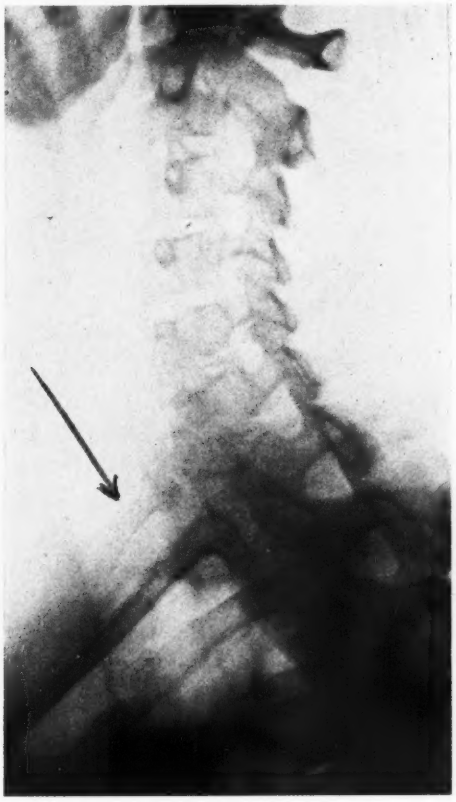


Fig. 7. Small cervical rib, not visible in anteroposterior or lateral views, but shown only in this oblique projection. The depression of the fractured first rib shown in Figure 6, to which this cervical rib is undoubtedly attached, produced symptoms of pain along the distribution of the brachial plexus. Illustrates the importance of special technical procedures directed by a roentgenologist familiar with the significance of clinical symptoms.

Most of the cases in this group have had only two views and frequently only a single view, and many of these have to be re-examined. In a recent case, the reports stated that a lumbar vertebra was fractured and the film came with a report to prove this diagnosis. The medical referee was not certain, while the roentgenologist

was almost certain that the crack in the vertebra was a gas shadow in the overlying colon. The man was ordered re-examined:

Fig. 8. Conditions in the lower dorsal spine, found five months after injury, the effects of which were supposed to be confined to the pelvis. Failure to secure films of the spine soon after the injury made it impossible to determine whether or not these changes were due to the injury.

the body cast was removed, and the film showed that the overlying gas bubble had moved and the fracture was "gone with the wind."

A very graphic illustration of the possible seriousness of faulty technic was that of a man who fell and bruised his hip. No x-ray examination was made as the man could walk. Some weeks later another surgeon examined him on account of persistent pain, making a film of the hip but with the thigh in external rotation—certain sign of an amateur. Diagnosis of an old impacted fracture of the femoral neck was made and the man put in a body cast. When the film came to the medical referee, the roentgenologist said there was no fracture. To prove the point a film was made of a normal pelvis, with one thigh rotated outward and the other inward. The surgeon treating the man was then requested to remove his cast and have a stereoroentgenogram made with the thigh in internal rotation. This was done and normal femoral neck was shown. Had the first film not been sent to the medical referee for inspection of consulting roentgenologist, this man would have been kept in his body cast for two months, as the surgeon's report stated that was his plan of treatment.

A man with a spine injury which crushed his eighth dorsal vertebra was properly treated and came up for final rating before the Advisory Council because his symptoms seemed greater than the injury would account for. A more comprehensive examination than any which had previously been made showed a fracture of the eleventh dorsal vertebra also.

(*c*) A third group of cases show bone changes or evidences of injury of questionable relation to the recent accident. It is this type of injury that gives the automobile insurance racketeers their opportunity. These criminals fabricate an automobile accident, claim injury to a part which is known to show bone abnormalities, secure x-ray films from some sympathetic surgeon inexperienced in x-ray appearances, or from some irregular practitioner, improperly though legally permitted to take x-ray films. With these, they scare the insurance company into settlement, and move on to a new field of activity. The racket has invaded the industrial field.

A man had an injury to his pelvis which was x-rayed and found negative. He was treated and discharged by his surgeon. Five months later he turned up and claimed that his back still hurt him, now locating the pain high up. This was examined and old changes found in the lower dorsal vertebræ (Fig. 8). He claimed his back had been perfectly normal until the injury five months previously, and while the Advisory Council had grave doubt about these changes having been produced by the injury in question, they were forced to give him the benefit of all doubt and rated him as 15 per cent permanently

disabled. A thorough examination in the outset might have prevented this possible error.

Fig. 9. Anomaly in development of second lumbar vertebra. This was found at examination, made the day after the accident. It was re-examined at intervals during the next eight months and no visible change could be detected. Medical Advisory Board decided it was a developmental anomaly, a decision which was made possible because of the prompt examination at time of injury.

In another case a deformed second lumbar vertebra was found in the spine of a young man injured by a fall from a considerable height at Boulder Dam (Fig. 9). The appearance of the vertebra never changed during the months following, and the final conclusion at the end of six months was that this deformity was not caused by the fall, but was either a congenitally deformed bone, or an old injury which the patient either could not recall or would not admit.

A recent report came in with a film, showing multiple fractures of the transverse processes of lumbar vertebræ (Fig. 10), which the surgeon thought must have been

Fig. 10. Film made immediately after a back injury on a patient who denied previous injury to the back. Surgeon reported the fractures as recent and produced by the injury under treatment. The Medical Referee and the consulting roentgenologist decided they were old and due to some prior injury.

produced by the accident, as the patient denied prior injury. However, the roentgenologist's opinion was positive that these fractures were months and probably years old.

In a wrist injury where the man claimed no previous accident, an old fractured scaphoid was found. During the period of observation, we accidentally ran across some old films of this man's wrist made in 1931. They showed the same condition exactly as it appeared five years later (Fig. 11). We have had quite a number of old Kienbock and Preiser lesions in the wrist, where the workmen denied prior injury.

(*d*) In the fourth group are included those cases showing pathological bone formations which are mistakenly interpreted as related to certain injuries.

In two instances, cases which probably do not belong strictly in this group, the interosseous ridge of the tibia has been

Fig. 11. Old divided scaphoid, found in a man who denied previous wrist injury. While under observation and compensation, an old film, taken five years previously, came to light which showed this identical condition then present.

interpreted as a traumatic periosteal proliferation, the interpretation being changed as time brought no change in the shadow.

One of the most interesting cases in this group was one where a triangular fragment of bone above the head of femur was interpreted as a fragment broken off the rim of acetabulum (Fig. 12). Persistent symptoms seemed to call for its removal, and a competent orthopedic surgeon searched diligently for the fragment, even exploring the hip joint. It was not found and subsequent examination showed the same density. The final conclusion was that it must be in the capsular ligament. This fragment resembles the os acetabuli which we have

Fig. 12. Bony formation at the upper margin of the acetabulum, diagnosed as a fragment detached from the pelvic bone and causing pain. Attempt at removal failed, as it could not be found. Subsequent examination showed the same shadow and final decision was that it is probably a calcification in the capsular ligament and of no clinical significance.

once or twice seen interpreted as fracture of the rim of acetabulum.

Two or three times, we have seen calcifications about the elbow joint interpreted as fractures or as associated with neuritis. One of these is still a live issue. The attending surgeon recommends its removal, and the roentgenologist is unable to agree that it is the cause of ulnar neuritis.

An interesting case recently was that of a cowboy who was thrown from his horse and found with a piece of bone protruding from the back of his thigh. Films were made and condition diagnosed as a compound fracture of shaft of femur, by the surgeon. Cast was applied. Abscess of soft tissues developed, and when this was incised several more pieces of bone came

away. When the abscess healed, films showed the femur to be intact. This was evidently a fracture of an osteoma which was finally eroded away completely by the infection.

We must not lose sight of the fact that infections and bone changes may follow injury and may not be shown by the first examination. Among the conditions of this type for which the roentgenologist must be on the watch is acute traumatic osteoporosis, the so-called Sudeck's atrophy. Since this may not develop for several weeks after injury, it is a serious mistake to consider a first negative report as final for that injury. The same caution applies in spine injuries, in which a Kummell deformity may develop when nothing is shown by the first examination. This is all in line with our theme that the roentgenologist should be a permanent and constant consultant in all industrial injuries.

Having given consideration to the causes and symptoms, we can make a diagnosis of the disease, pseudo-roentgenology, afflicting our specialty and we should be able to outline the treatment. As in all medical problems, treatment is the thing we are most interested in.

Are the illustrations here presented designed to promulgate the idea that the suffusion throughout our medical organism of small x-ray plants is an unhealthy condition and should be combated? Not necessarily so. The leucocytosis resulting from infection is very desirable, but if it changes to a leukemia, that is another matter. The general use of x-ray in diagnosis has contributed to medical progress and can contribute still further, but it should be held within the bounds of health. That is our problem.

The most progressive step we have made in a generation was the organization of The American Board of Radiology, whose function is to determine and specify who shall be entitled to be classed as specialists in roentgenologic interpretation. We cannot, of course, expect to have these designations accepted by the public or public servants until the medical profession itself has accepted them. It is not sufficient that the American Medical Association accepts the Board's classification for its medical directory. It must go much further than this. The Council on Medical Education and Hospitals should lay down as a definite essential for hospitals approved for intern training that the department of diagnostic roentgenology, as well as all radiotherapy, shall be under the supervision and direction of diplomates of the Board of Radiology.

Having accomplished this step, we can go to Industrial Commissions and insurance carriers with the demand that competent roentgenologists shall review their cases and personally supervise all difficult examinations. Also that only adequate and proper examinations are entitled to be paid for.

The courts will be the hardest to deal with, because institutions which have not changed their ideas or procedures in a thousand years find it difficult to keep up with rapidly moving developments. Occasionally, we see a flash of intelligence from a jurist in the matter of x-ray examinations, as in the case of one Federal judge who decreed that x-ray films were not needed, so long as the roentgenologist could tell what was shown by them. Wise judge, may his tribe increase!

In summary, then, our contention is that only those who are expert shall pretend to be so. We have several excellent precedents for this claim. The College of Surgeons has been working for years to prevent incompetent surgeons from doing major surgery. Even though the American Board of Pathology is just being formed, we would be astounded if a doctor whose only qualification was the possession of a microtome and a microscope should attempt to make tissue diagnoses, and yet exactly that ridiculous situation holds in the field of diagnostic roentgenology. Many practitioners carry ophthalmoscopes to observe gross conditions in the eyes, but their opinions would not be accepted in court as against those of ophthalmolo-

gists. Illustrations might be multiplied *ad infinitum*.

If the hospitals approved by the Council on Education for intern training, and those certified by the American College of Surgeons, were required to have their diagnostic x-ray work directed by approved roentgenologists, if Industrial Commissions and insurance carriers would accept as conclusive only the findings of such experts, if the courts would hold as of value on these matters the opinions only of those recognized as competent by the bodies qualified to pass on them, we will have gone a long way toward stopping the progress of this disease which is now parasitically flourishing on the fair body of our specialty.

EVACUATION OF THE GALL BLADDER IN PEPTIC ULCER PATIENTS[1]

By EDWARD A. BOYDEN, PH.D., *and* THEODORE M. BERMAN, M.D., *Minneapolis, Minn.*

From the Departments of Anatomy and Radiology, University of Minnesota[2]

IN considering extra-biliary lesions that might affect the normal function of the bile passages, one naturally turns first to erosions of the stomach and duodenum, partly because the intake of food is so closely associated with expulsion of bile and partly because it has been suspected, since the time of Claude Bernard (1), that hydrochloric acid plays some rôle in regulating the flow of bile. Furthermore, these lesions vary among themselves, it being now generally recognized that peptic ulcer is characterized by an abundance of free acid whereas carcinoma of the stomach and pernicious anemia are distinguished, respectively, by a decrease and virtual suppression of free acid. Here then, would seem to be an experiment provided by Nature for testing the rôle played by gastric secretion.

To Bronner (2) must go the credit for first pointing out that in peptic ulcer patients the gall bladder empties rapidly. He considered, indeed, that the gall bladder of his ulcer cases exhibited greater motility than that of any normal group of the population. He even went so far as to predict that ulcer could be diagnosed by the rate of emptying of the gall bladder. Since that time, however, more detailed information has appeared concerning the normal rate of emptying in different sex and age groups. We know, now, that individual variation is so great that little can be predicted from a single case. It is only when we obtain the mean curves of evacuation in a relatively large number of individuals that we can say, for instance, that, on the average, the gall bladders of boys empty much faster than those of girls (3), that after puberty the male gall bladder empties significantly slower than the female (4), and that in old age (5) there is, at least, no diminution in rate of emptying in either sex.

Bronner, however, drew his conclusions from just 12 peptic ulcer cases (four women and eight men) and half of these were complicated by short or long standing gastroenterostomies. Furthermore, his estimates of gall-bladder volumes were based on purely visual inspection of cholecystograms. It has seemed desirable, therefore, to restudy this problem with the aid of quantitative methods and more adequate controls.

To test the hypothesis outlined above, we have employed the same technic that was used in studying normal individuals. Following visualization of the gall bladder by oral administration of "iodeikon," cholecystograms were taken at 2, 4, 8, 12, 16, 20, 25, 30, 35, 40, and 45 minutes after a standard meal of four egg yolks, the latter well mixed in an equal volume of milk, seasoned with a pinch of sugar and a few drops of vanilla. The outlines of the gall bladder were then transferred to tracing paper and divided into segments representing circular plinths. From these the changing volumes of the gall bladder were computed according to the methods described in preceding articles (3, 4).

Incidentally, ulcer patients were studied in the order they appeared for diagnosis in the Department of Radiology and before treatment for ulcer had been instituted. Altogether, cholecystograms were made from 33 male patients; of these, 10 series

[1] Read by Dr. Boyden before the Radiological Society of North America at the Twenty-second Annual Meeting, at Cincinnati, Nov. 30–Dec. 4, 1936.

[2] Aided by grants from the Medical Research Fund of the Graduate School. The senior author also wishes to express appreciation to Dr. L. G. Rigler for the facilities generously provided by the Department of Radiology.

were discarded, three because of lack of visualization of the gall bladder, six because the series was incomplete (through gastric ulcer group empty much faster than those of normal males (3, 5), *i.e.*, the former have discharged more than

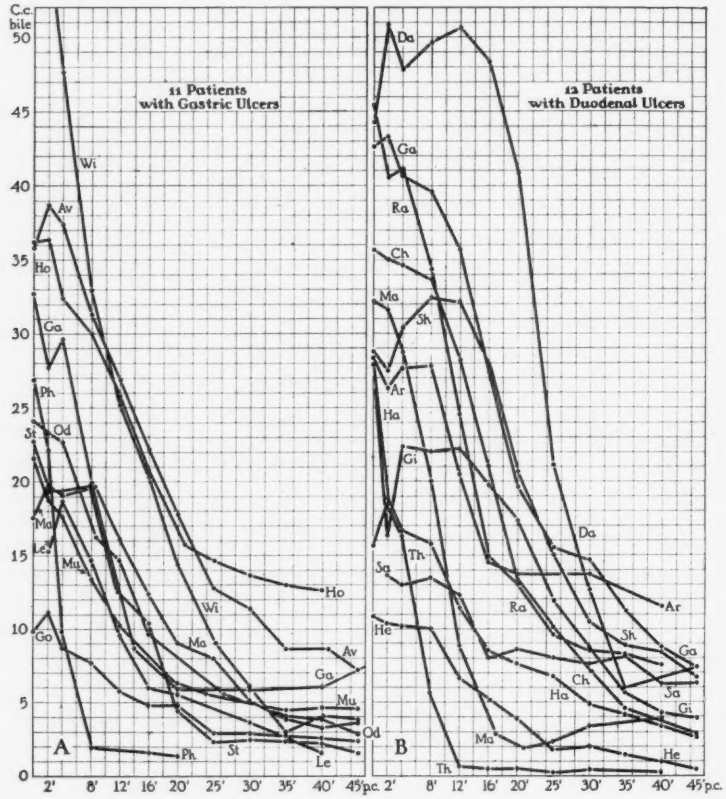


Fig. 1. Individual curves of contraction of the gall bladder in 23 peptic ulcer patients of the University Hospital: *c. c. bile*, volume of gall bladder in cubic centimeters; *0–45' p. c.*, minutes after standard meal of 4 egg yolks; letters represent names of patients (*cf.* Table I).

faintness of shadows or obliteration due to gas, stools, barium, etc.), and one because the gall bladder failed to empty at all. The remaining 23 (11 gastric ulcer series and 12 duodenal ulcer series) are listed in Table I (*A* and *B*, respectively) and form the basis of this report. The individual curves of evacuation will be found in Figure 1. By translating these into terms of percentage loss of volume in given units of time, the mean curves of evacuation for the different groups have been obtained. These are shown in Figure 2.

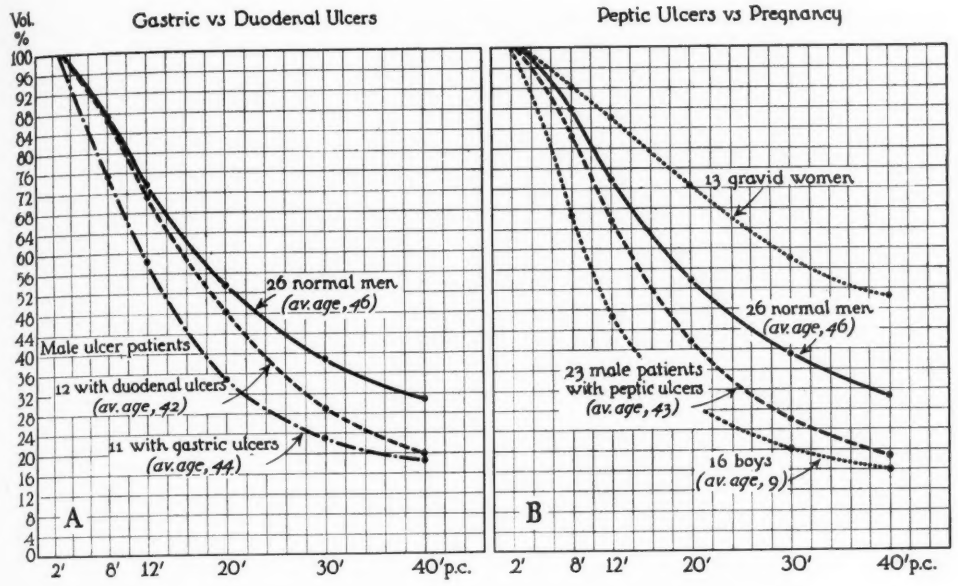
Inspection of Figure 2-*A* shows that on the average, gall bladders of the male four-fifths, the latter only approximately two-thirds, of their contents at the end of the first 40 minutes. When subjected to statistical analysis, these differences are found to be highly significant, *i.e.*, the probability (P × 100) that these differences may be due to random sampling is less than four in a thousand (see last column, Table II-*C*). So also at 12, 20, and 30 minutes post-cibum the differences are significant, the probability ranging from 4.6 in a hundred to 5 in ten thousand.[3]

[3] In ordinary statistical parlance, if a difference occurs less than five times in a hundred it is assumed that it is

TABLE I.—EMPTYING OF GALL BLADDER IN PEPTIC ULCER PATIENTS

		Weight, lbs.		Ulcer			Gastric Retention	HCl Acid		Volume of Gall Bladder at Intervals after Standard Meal											
										2 min.	8 min.		12 min.		20 min.		30 min.		40 min.	
A. 11 Gastric Ulcer Patients	Age	Normal	Loss	Location[1]	Duration		Free	Total	c.cm.	c.cm.	Percentage Loss	c.cm.	Percentage Loss	c.cm	Percentage Loss	c.cm.	Percentage Loss	c.cm.	Percentage Loss
E. Av.	56	135	2	above incisura	15 yrs.	none	16°	29°	37.39 (4 min.)	31.22	16.50	26.90	28.06	17.84	52.29	11.42	69.46	8.66	76.84
E. Ga.	33	120	...	below cardia	1 yr.	none	....	...	27.84	19.95	28.34	12.50	55.10	5.88	78.88	5.88	78.88	(6.03)	78.88
E. Go.	47	135	9	high up[2]	2 yrs.	20%	much	...	11.15	7.74	30.55	5.78	48.20	4.86	56.45	2.89	74.10	2.62	76.47
J. Ho.	28	180	5	above incisura	3 yrs.	none	18°	33°	32.41 (4 min.)	30.05	7.93	25.85	20.25	16.60	48.78	13.65	57.89	12.70	60.82
A. Le.	55	120	10	above incisura	7 mos.	none	43°	54°	15.22	14.71	3.34	9.58	37.08	5.78	62.05	3.67	75.87	1.58	89.65
K. Ma.	56	135	7	post. wall[2]	6 wks.	none	present	...	17.45 (0 min.)	(19.68)	00.00	16.14	7.53	9.06	48.10	5.12	70.66	3.28	81.20
O. Mu.	36	180	38	above incisura	8 mos.	none	38°	44°	18.76	13.38	28.68	10.23	45.47	6.30	66.42	5.04	73.14	4.59	75.54
H. Od.	29	165	20	middle third[3]	2 yrs.	none	present	...	22.57 (4 min.)	16.95	24.90	14.17	37.23	8.00	64.56	4.98	77.92	3.94	82.56
L. Ph.	51	150	18	middle third[4]	2 yrs.	none	....	...	22.04	1.97	91.07	1.77	91.96	1.44	93.47	1.44	93.47	1.44	93.47
D. St.	51	140	17	at incisura	22 mos.	5%	54°	69°	19.42	(19.81)	00.00	12.99	33.09	5.30	72.70	2.50	87.85	2.23	88.50
G. Wi.	43	170	20	middle third[5]	3 yrs.	none[5]	51°	66°	54.32	32.93	39.38	25.19	53.63	14.30	73.67	6.03	88.90	(2.89)	94.68
	485	1630	146				220	295	278.57		270.69		457.60		717.37		848.14		898.61
Mean	44.1	148.2	14.6				36.7	49.2	25.32		24.61		41.6		65.22		77.10		81.69
B. 12 Duod. Ulcer Patients				Niche															
W. Ar.	55	175	10	large[6]	3 mos.	none	52°	42°	26.24	(27.82)	00.00	20.46	22.01	13.78	47.50	13.78	47.50	11.54	56.01
L. Ch.	40	160	none	large	1.5 yrs. 3 mos.	some at 6°	....	...	33.60 (8 min.)	33.60	00.00	28.21	16.05	13.51	59.79	7.30	78.27	3.75	88.84
J. Da.	53	165	7	large	20 yrs. 10 days	none	48°	48°	44.34 (0 min.)	(49.73)	00.00	(50.64)	00.00	40.94	7.69	12.59	71.60	5.64	87.28
L. Ga.[7]	46	135	35	yes	7 mos.	none	49°	58°	40.80 (4 min.)	39.62	2.88	35.69	12.53	21.30	47.80	14.70	63.98	8.53	79.10
E. Gi.	68	151	10	yes	4 wks.	none	14°	27°	22.18 (4 min.)	(22.04)	00.00	(22.18)	00.00	17.32	21.90	8.79	60.35	4.2	81.06
M. Ha.	61	121	37	duod. def.	2 yrs. 3 mos.	.....	none	27°	18.90	15.74	16.68	11.42	39.59	7.74	59.02	4.86	74.30	3.41	81.96
E. He.	25	142	17	duod. def.	6 yrs.	none[8]	....	...	10.37	9.06	12.65	6.56	36.73	3.94	62.04	1.99	82.25	1.05	89.89
M. Ma.[9]	33	185	15	yes	13 yrs.	none	35°	40°	31.62	20.07	36.51	8.79	72.19	2.2	93.04	(3.41)	93.78	(3.81)	93.78
C. Ra.	38	172	5	duod. def.	1 yr.?	none	44°	50°	39.49	34.38	12.95	24.54	37.86	13.25	66.45	8.66	78.08	7.48	81.06
W. Sa.	24	165	8	yes	5 yrs.	none	52°	56°	12.99 (4 min.)	(13.51)	00.00	12.34	5.05	8.53	38.42	7.48	42.43	6.17	52.52
A. Sh.	29	188	26	yes	4 yrs.	10%	15°	24°	27.55	(32.41)	00.00	(32.14)	00.00	21.65	21.43	10.50	61.90	8.4	69.51
W. Th.	38	148	17	large	3 mos.	none	35°	43°	16.53 (4 min.)	5.64	65.88	0.66	96.03	0.53	96.81	0.26	98.40	0.26	98.40
	510	1907	187				344	415	324.61		147.55		338.04		621.89		852.84		959.41
Mean	42.5	159	15.6				34.4°	41.5	27.05		12.30		28.17		51.82		71.07		79.95

[1] On lesser curvature except in patient "Ma." [2] Also deformity of duodenal bulb. [3] Also duodenal niche, but gastric ulcer was cause of acute symptoms; attacks of colic localized in right upper quadrant; hypermotile bowel; head of meal in splenic flexure at 5 hours. [4] Penetrating type, also duodenal deformity. [5] Duodenal deformity; patient subsequently developed carcinoma of stomach with disappearance of free acid and appearance of 25 per cent retention. [6] Radiation of pain to scapula and hypochondrium suggesting peritoneal inflammation. [7] Gastro-enterostomy, 8 years previously. [8] Fifty per cent retention a week before and 14 months after cholecystography. [9] Gastro-enterostomy 11 years previously.


Vol. %
Gastric vs Duodenal Ulcers
Peptic Ulcers vs Pregnancy
26 normal men (av. age, 46)
Male ulcer patients
12 with duodenal ulcers (av. age, 42)
11 with gastric ulcers (av. age, 44)
13 gravid women
26 normal men (av. age, 46)
23 male patients with peptic ulcers (av. age, 43)
16 boys (av. age, 9)
A
B
2' 8' 12' 20' 30' 40' p.c.

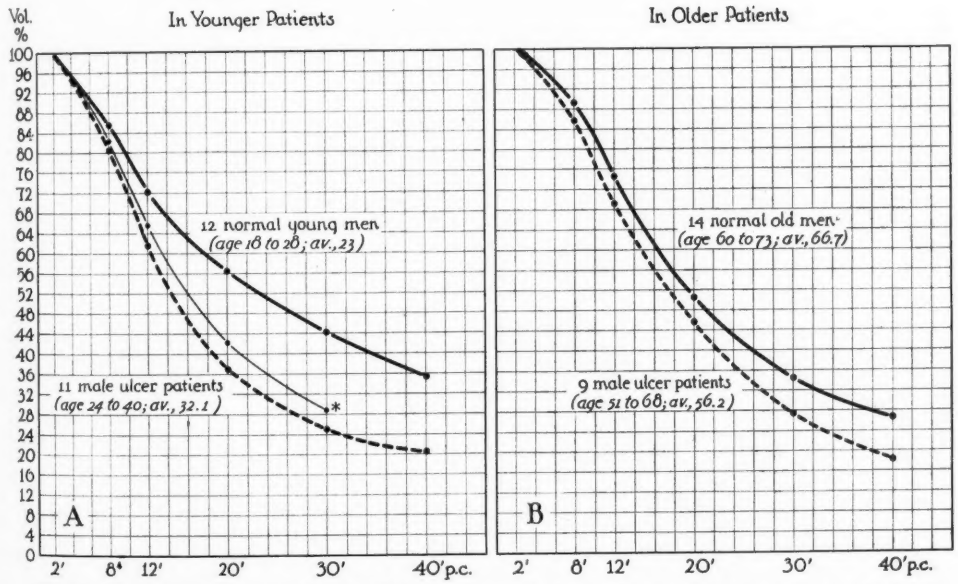

Vol. %
In Younger Patients
In Older Patients
12 normal young men (age 18 to 28; av., 23)
11 male ulcer patients (age 24 to 40; av., 32.1)
14 normal old men (age 60 to 73; av., 66.7)
9 male ulcer patients (age 51 to 68; av., 56.2)
A
B
2' 8' 12' 20' 30' 40' p.c.

Fig. 2. Mean curves of evacuation of gall bladder in peptic ulcer patients, reduced to percentage basis (*cf.* Table I). *Vol. %*, mean percentage of bile in gall bladders of a given group at a given time. *A*, gastric and duodenal ulcer curves; *B*, combined peptic ulcer data contrasted with the norm for men (Boyden and Grantham, 1936), with the curve for gravid women (Gerdes and Boyden, 1936) and with the curve for young boys (Boyden and Fuller, 1934). (*Upper curves.*)

Fig. 3. Mean curves of evacuation of gall bladder in peptic ulcer patients separated into two groups on the basis of age. *A*, young ulcer patients contrasted with the norm for young men. *Asterisk* indicates curve for nine normal young men (*cf.* Table III) who were given one egg yolk by duodenal tube (compiled from data of Boyden and Birch, 1930). *B*, older patients contrasted with the norm for older men. (*Lower curves.*)

not due to chance. As in preceding papers the writer is indebted to Dr. Edith Boyd, of the Department of Anatomy, for supervising the statistical analyses.

The duodenal ulcer curve, on the other hand, is intermediate in position, and its

TABLE II.—STATISTICAL ANALYSES

Time Interval (min.)	No. of Cases	Mean and Standard Error	No. of Cases	Mean and Standard Error	Difference and Standard Error	Diff. S. E.	Probability Integral
		A. Gastric	*vs.*	Duodenal Ulcer Patients			
8	11	24.61 ± 7.81	12	12.30 ± 5.82	12.31 ± 9.74	1.26	0.208
12	11	41.60 ± 6.63	12	28.17 ± 8.78	13.43 ± 11.00	1.22	0.222
20	11	65.22 ± 4.18	12	51.82 ± 7.85	13.40 ± 8.89	1.51	0.131
30	11	77.10 ± 3.01	12	71.07 ± 4.87	6.03 ± 5.73	1.05	0.293
40	11	81.69 ± 2.94	12	79.95 ± 4.09	1.74 ± 5.04	0.35	0.727
		B. Ulcer Patients	*vs.*	Normal Males			
8	23	18.18 ± 4.88	26	12.12 ± 2.16	6.06 ± 5.34	1.13	0.258
12	23	34.59 ± 5.63	26	26.59 ± 3.50	8.00 ± 6.63	1.21	0.226
20	23	58.23 ± 4.68	26	46.62 ± 4.56	11.61 ± 6.53	1.78	0.075
30	23	73.96 ± 2.93	26	60.83 ± 3.51	13.13 ± 4.57	2.87	0.0037
40	23	80.78 ± 2.51	26	69.30 ± 3.02	11.48 ± 3.93	2.92	0.0037
		C. Gastric Ulcer Patients	*vs.*	Normal Males			
8	11	24.61 ± 7.81	26	12.12 ± 2.16	12.49 ± 8.10	1.54	0.124
12	11	41.60 ± 6.63	26	26.59 ± 3.50	15.01 ± 7.50	2.00	0.046
20	11	65.22 ± 4.18	26	46.62 ± 4.56	18.60 ± 6.19	3.00	0.0027
30	11	77.10 ± 3.01	26	60.83 ± 3.51	16.27 ± 4.62	3.52	0.00050
40	11	81.69 ± 2.94	26	69.30 ± 3.02	12.39 ± 4.21	2.94	0.0037
		D. Duod. Ulcer Patients	*vs.*	Normal Males			
8	12	12.30 ± 5.82	26	12.12 ± 2.16	0.08 ± 6.21	0.01	0.992
12	12	28.17 ± 8.78	26	26.59 ± 3.50	1.58 ± 9.45	0.17	0.865
20	12	51.82 ± 7.85	26	46.62 ± 4.56	5.20 ± 9.08	0.57	0.569
30	12	71.07 ± 4.87	26	60.83 ± 3.51	10.24 ± 6.00	1.71	0.087
40	12	79.95 ± 4.09	26	69.30 ± 3.02	10.65 ± 5.08	2.10	0.036
		E. Young Ulcer Patients	*vs.*	Normal Young Males			
8	11	19.80 ± 6.03	12	14.3 ± 3.37	5.50 ± 6.91	0.80	0.424
12	11	38.36 ± 8.64	12	28.0 ± 5.77	10.36 ± 10.39	1.00	0.317
20	11	63.33 ± 6.65	12	43.5 ± 7.59	19.83 ± 10.09	1.97	0.049
30	11	74.81 ± 4.79	12	55.7 ± 5.43	19.11 ± 7.24	2.64	0.0093
40	11	79.25 ± 4.23	12	64.6 ± 4.75	14.65 ± 6.36	2.30	0.021
		F. Old Ulcer Patients	*vs.*	Normal Old Males			
8	9	14.18 ± 9.89	14	10.29 ± 2.78	3.89 ± 10.27	0.38	0.704
12	9	28.81 ± 9.40	14	25.42 ± 4.39	3.39 ± 10.37	0.33	0.741
20	9	51.64 ± 8.50	14	49.29 ± 5.60	2.35 ± 10.18	0.23	0.818
30	9	72.34 ± 4.53	14	65.25 ± 4.37	7.09 ± 6.29	1.13	0.258
40	9	81.77 ± 3.67	14	73.27 ± 3.78	8.50 ± 5.27	1.61	0.108
		G. Normal Males (5 yolks by mouth)	*vs.*	Normal Males (1 yolk by tube)			
8	12	14.26 ± 3.38	9	17.87 ± 6.49	−3.61 ± 7.32	.49	0.624
12	12	27.96 ± 5.79	9	34.28 ± 7.42	−6.32 ± 9.41	.67	0.503
20	12	43.51 ± 7.59	9	57.62 ± 5.04	−14.11 ± 9.10	1.55	0.121
30	12	55.67 ± 5.46	9	70.99 ± 3.90	15.32 ± 6.71	2.28	0.023
40	12	64.55 ± 4.81	..	.....	.....	..	...

differences from the norm are significant only at the forty-minute reading (Table II-*D*). When, however, both ulcer groups are combined (Fig. 2-*B*) the curve for ulcers is significantly steeper than that for the norm in the 20-, 30-, and 40-minute periods (Table II-*B*). It is not so steep as the curve for young boys, although the two resemble each other closely in shape. By way of contrast, and to indicate the other extreme in rate of evacuation, we have added to Figure 2-*B* the curve for gravid women (6).

At this point objection may be raised to the fact that the norm is made up of two groups—a combination of 12 young men (age 18 to 28) and 14 older men (age 60 to 73); whereas the 23 men in the ulcer group range all the way from 24 to 68 years of age. Their distribution according to decades is as follows:

	20–39	30–40	41–49	50–59	60–69
11 Gastric ulcer patients	2	2	2	5	0
12 Duodenal ulcer patients	3	4	1	2	2
Total	5	6	3	7	2
	Group 1			Group 2	

With this grouping as a basis we have made a new set of comparisons (Fig. 3-*A*), placing the mean curve of emptying of Group 1 (made up of 11 ulcer patients below the age level of the climacteric) against the curve of 12 normal young men. When these curves are analyzed statistically, the differences are found to be significant in the 20-, 30-, and 40-minute readings (Table II-*E*). Thus it is clear that in younger men, peptic ulcer markedly accelerates the discharge of bile from the gall bladder.

When we turn to the men above the age level of the climacteric (Fig. 3-*B*) the difference between ulcer patients and the norm is not marked enough to be statistically significant (Table II-*F*). This may mean exactly that which the curves suggest, namely, that erosions of the stomach and duodenum in older groups have less effect on the discharge of bile than in younger groups. Of interest in this connection are the studies of Sagal, Marks, and Kantor (7), who have shown that gastric acidity is highest in the earlier age groups, reaching its peak in the fifth decade, and lowest in later decades. Even if free acid is not the cause of accelerated discharge of bile from the gall bladder, decrease in acid in old age may reflect a decrease in the factor which does regulate the rate of emptying and so afford a clue as to why the differences between the ulcer curve and the norm of old age are not greater.

On the other hand, the normal curves for old men and women, as pointed out in a previous study (5), are not so satisfactory as those for young men and women, because in the latter group, made up chiefly of medical students, 100 per cent of the gall bladders were visualized, whereas in the former (made up of hospital patients without obvious biliary or gastro-intestinal histories) only 76 per cent of those studied could be visualized. So the conservative position was taken that although the gall bladder of old adults was on the borderline of being significantly faster than that of young adults, it was merely because we were dealing with the more favored members of an old age group, namely, those whose gall bladders were capable of being visualized. Probably, therefore, our normal curve of emptying for old age is too fast. Bearing this in mind, as well as the fact that in Figure 3-*B* the ulcer patients average ten years younger than those constituting the "normal" old age group, together with the further observation that all the ulcer curves are consistently in the direction of being faster, we have little hesitation in concluding that the effect of peptic ulcer even in older men is to increase the rate of discharge of bile from the gall bladder.

DISCUSSION

Granting that the above finding is true, what clinical significance has it? Probably none, for a somewhat more rapid flow of concentrated bile after meals

would seem to have no physiological significance, especially since such a rate of flow would be within the limits of normal variation. Its value then is chiefly negative; it strongly suggests that one does not need to fear that peptic ulcer will lay the foundation for biliary disturbances. This, of course, is in marked contrast to the prognosis which prevails in pregnancy.

However, the fact that the influence of ulcerations on the biliary tract is benign does not render it any less important, because it may eventually throw light on the mechanism of biliary disease. What factors, then, are responsible for this acceleration of flow? Does the size of the ulcer, for instance, make any difference? Apparently there is no obligatory relation between the two, for if we compare Figures 4 and 5 we see two craters of the same size (4-*A* and 5-*A*), the associated gall bladders of which empty at greatly differing rates (4-*B* and 5-*B*). So with the duodenal niches (Figs. 6 and 7). In this connection, however, it may be noted that in a larger series Vanzant, *et al.*, have reported that the acidity varies with the size of the ulcer (8).

Secondly, does the increased motility of the stomach play any rôle, either through reflex stimulation of the gall bladder and sphincter of Oddi, or through the initial delivery into the duodenum of larger volumes of hormone-provoking food? Superficial studies would suggest a negative answer, for it is not difficult to find individuals in whom a rapidly emptying gall bladder is associated with an eight-hour stomach or with a sluggish intestinal tract that requires 120 hours for the passage of a barium meal (9). Nevertheless, the more one deals with such factors the

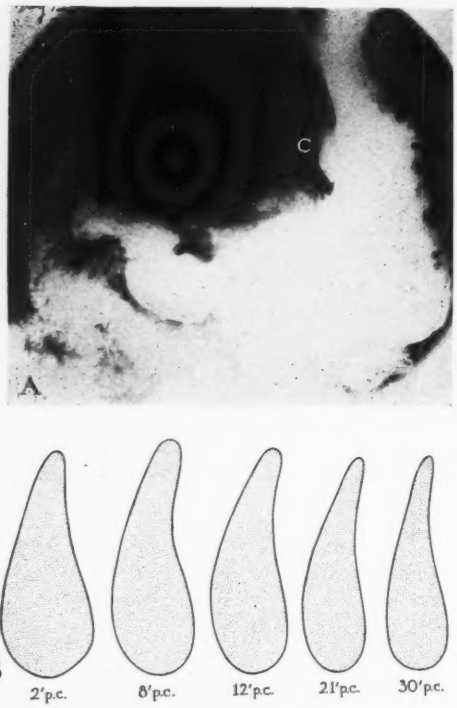


Fig. 4. Case *Ho.* (Table I-*A* and Fig. 1-*A*.) *A*, roentgenogram of stomach. (*C* = crater of large gastric ulcer); *B*, tracings of selected cholecystograms, showing slow evacuation of gall bladder.

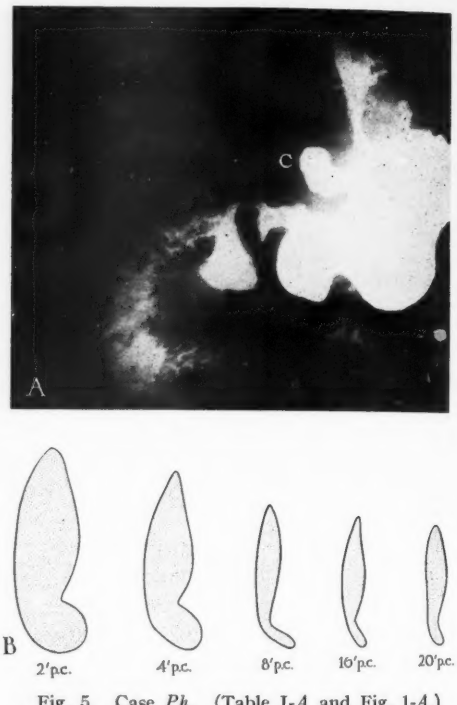
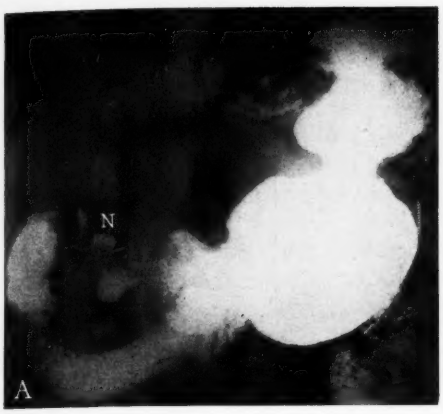


Fig. 5. Case *Ph.* (Table I-*A* and Fig. 1-*A*.) *A*, roentgenogram of stomach (*C* = large perforating ulcer); *B*, tracings of cholecystograms, showing fast emptying of gall bladder.

more he suspects that their existence cannot be ruled out until the mean value has been obtained in a larger number of cases, has yet determined the minimum amount of egg-yolk in the duodenum necessary to produce a maximum phase of contrac-



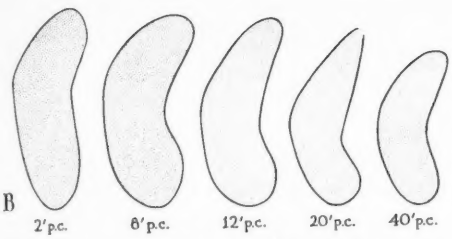
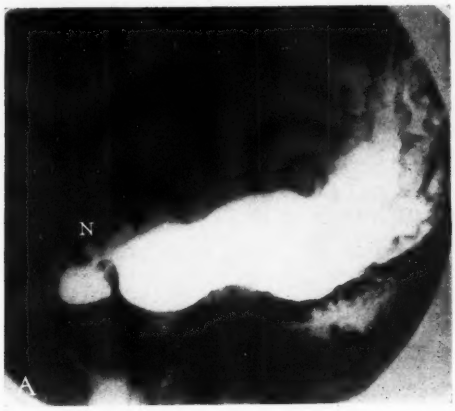


Fig. 6. Case *Ar*. (Table I-*B* and Fig. 1-*B*.) *A*, roentgenogram of stomach and duodenum (*N* = niche of duodenal ulcer); *B*, tracings of cholecystograms showing slow emptying of gall bladder.



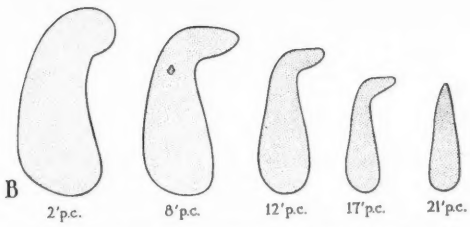


Fig. 7. Case *Ma*. (Table I-*B* and Fig. 1-*B*.) *A*, roentgenogram of stomach and duodenal cap (*N* = niche of duodenal ulcer); *B*, tracings of cholecystograms, showing fast emptying of gall bladder.

probably because the biliary tract is regulated by a number of factors, any one of which may, upon occasion, be masked by another. However, the evidence is quite convincing that the human gall bladder, as contrasted with that of the cat (10), for instance, is not subject to reflexes originating in the stomach, for painful faradic stimulation applied through a Rehfuss tube causes neither contraction of a relaxed gall bladder nor inhibition of one that is evacuating after a fatty meal (11). Whatever effect is produced by increased motility of the stomach must, therefore, act in another way—perhaps through the discharge of larger quantities of food into the duodenum in the first half-hour after a meal.

So far as the writers are aware, no one tion of the gall bladder. It must be quite small, for we have repeatedly induced it by injecting just one egg-yolk (30 c.c. by volume, when mixed with water, 12). Incidentally, the mean curve of evacuation in nine such cases is significantly faster than in a comparable student group to whom four or five egg-yolks were given by mouth (Fig. 3-*A*, see *, and Table II-*G*). Therefore, it is quite probable that the tendency of the stomach in peptic ulcer patients to discharge its contents with an initial rush—as Dr. Rigler informs the writers is usually the case—is an important factor in accelerating the discharge of bile.

In this sense, but not in the sense of Bronner (2), who considered that the vagus acted directly on the gall bladder of

TABLE III.—EMPTYING OF GALL BLADDER IN NINE NORMAL YOUNG MEN AFTER INJECTION OF ONE EGG-YOLK INTO DUODENUM

Name	Age	Wt.	Volume of Gall Bladder at 2 min. c.cm.	8 min. c.cm.	8 min. Percentage Loss	12 min. c.cm.	12 min. Percentage Loss	20 min. c.cm.	20 min. Percentage Loss	30 min. c.cm.	30 min. Percentage Loss
W. F. (Fig. 3*)	20	115	25.8	18.2	29.50	14.8	42.64	10.3	60.00	10.30	60.00
M. K. (Fig. 2*)	21	128	28.0	27.6	1.43	22.2	30.57	10.35	63.00	7.40	73.57
G. A. K. (Fig. 1†)	30	130	32.61 (4 pc)	17.56	46.14	11.21	65.62	11.07	66.06	7.52	78.28
E. D. P. (Fig. 3*)	23	140	19.63	19.26	1.88	18.89	3.77	15.00	23.58	9.45	51.86
A. R. (Fig. 1†)	16	165	68.76 (4 pc)	67.86	1.31	60.80	11.58	35.55	48.30	25.11	63.48
M. M. S. (Fig. 3*)	22	130	20.96	19.93	5.10	15.94	24.10	9.30	55.71	2.51	88.05
T. J. W. (Fig. 3*)	21	154	22.58	14.31	33.63	10.62	52.97	9.00	60.14	8.16	63.87
A. D. W.(1) (Fig. 1*)	21	149	46.08	26.82	41.80	18.20	60.51	9.90	78.52	(19.92)	80.08
A. D. W.(2) (Fig. 1*)	21	149	61.99 (4 pc)	(62.58)	00.00	51.60	16.77	22.80	63.23	12.60	79.68
	195	1260	326.41		160.79		308.53		518.54		638.87
Mean	22	140	36.27		17.87		34.28		57.62		70.98

* For individual curves of contraction, see *Boyden and Birch*, 1930.
† For curve, see *Boyden and Saunders*, 1928.

ulcer patients, increased flow of bile in the presence of peptic ulcer may be attributed to the fact that the patient is vagotonic. Of special interest in this connection is Babkin's observation that the gastric glands on the lesser curvature are more actively under the control of the vagus than those in the region of the greater curvature (13). Whether the human sphincter of Oddi, as contrasted with the occluding apparatus in mammals (14), is also subject to vagotony has yet to be proven but cannot be disregarded.

This brings us to our third query. To what extent is the discharge of bile influenced by increments of free acid from the stomach? As indicated in the reviews of the subject by Babkin (15) and Ivy (16), there is abundant evidence that in animals, dilute HCl introduced into the duodenum causes contraction of the gall bladder and a drop in pressure at the outlet of the common duct. But in man, our experience has been that in four out of eight attempts, duodenal injection of 20 to 40 c.c. of dilute acid ranging from 10° to 75° produced merely a small reduction of the gall-bladder shadows, lasting from 8 to 14 minutes only and never resulting in aspiration of B bile (12). In our present state of knowledge regarding the formation of gall-bladder hormones, what effect the acid would have when mixed with egg-yolk can only be conjectured. Furthermore, if the free acid were the critical factor in the acceleration, we would expect the curve of evacuation in duodenal ulcer patients to be faster than those with gastric ulcer—the reverse of the situation in Figure 2-*A*—for several authors (8, 17, 18) have recently shown that the highest acid values are associated with duodenal ulcer.[4]

On the other hand, the distinguishing feature of gastric secretion in man, as contrasted with the dog—in which animal it is produced only at maximum concentration (19)—is its great variability, both in concentration and in quantity. Thus Berglund, Johnson, and Chang (20) who have taken the position that the important measure of gastric secretion is not the free acid but the total output of chlorides,

[4] In our series the record of histamine tests indicates that the amount of free acid is approximately the same for gastric and duodenal ulcer groups (Table I). But these routine hospital reports cover only 16 patients and are otherwise incomplete. They are included to show that all but one of the patients tested had free acid and that the titer was within the range of what is considered normal.

have shown that the mean for gastric secretion in duodenal ulcer patients is only on the borderline of being significantly higher than the normal mean. The mean for achlorhydria and especially the mean for pernicious anemia, however, are significantly lower than the normal. In view of these various considerations it would be folly at this time to deny the possibility that free acid is an acceleratory factor in the evacuation of bile in peptic ulcer patients. On the other hand, it would seem that the effects of peptic ulcer could be explained without resort to the theory of stimulation of the duodenum by spurts of acid chyme. It is hoped that our impending study of pernicious anemia patients may afford another means of separating the acid from the food factor, for in such patients we may expect a steady initial flow of food from the stomach that would be unmixed with free acid. In this connection, it may be noted that our first returns on patients with carcinoma of the stomach (21) indicate that in this disease the gall bladder empties at a normal rate.

SUMMARY

(1) Visualization of the gall bladder (oral method) occurred in 30 out of 33 peptic ulcer patients.

(2) From this number, 23 good series of cholecystograms were obtained after a standard meal of four egg-yolks and milk.

(3) Computation of the changing volumes of the gall bladder has indicated that at least in men under forty, and probably in older men as well, peptic ulcer markedly increases the rate of evacuation of the gall bladder.

(4) In discussing the various factors that might be responsible for this accelerated discharge of bile, it was shown that when a single egg-yolk is placed directly into the duodenum the gall bladder empties significantly faster than when four or five egg-yolks are given by mouth.

(5) It is suggested, therefore, that the greater tonus of the stomach in peptic ulcer cases may be responsible for a greater initial rush of food into the duodenum, which, in turn, causes faster evacuation of the gall bladder. Possibly, also, this vagotony may relax the sphincter of Oddi.

(6) The theory that the large amounts of free acid in the stomach of peptic ulcer patients may also provoke the duodenum to greater production of cholecystokinin is withheld pending a survey of the rate of emptying of the gall bladder in achlorhydric patients.

REFERENCES

(1) BERNARD, CLAUDE: Leçons de physiologie expérimentale. 1856, **2**, 429. (Quoted by Babkin, 1928.)

(2) BRONNER, H.: Die cholecystographische Motilitaetspruefung der Gallenblase und ihre Ergebnisse (eine klinisch-roentgenologische Studie). Fortschr. a. d. Geb. d. Röntgenstrahlen, 1929, **39**, 23.

(3) BOYDEN, EDWARD A., and FULLER, ALICE H.: Anatomy and Physiology of the Gall Bladder in Children: A Cholecystographic Study. Am. Jour. Dis. Child., 1934, **48**, 565.

(4) BOYDEN, EDWARD A.: An Analysis of the Reaction of the Human Gall Bladder to Food. Anat. Rec., 1928, **40**, 147.

(5) BOYDEN, E. A., and GRANTHAM, S. A., JR.: Evacuation of the Gall Bladder in Old Age. Surg., Gynec. and Obst., 1936, **62**, 34.

(6) GERDES, MAUDE M., and BOYDEN, EDWARD A.: Retardation of the Gall Bladder in Pregnancy. Proc. Soc. Exp. Biol. and Med., December, 1936, **35**, 393.

(7) SAGAL, Z., MARKS, J. A., and KANTOR, J. L.: The Clinical Significance of Gastric Acidity. Ann. Int. Med., 1933, **7**, 76.

(8) VANZANT, F. R., ALVAREZ, W. C., BERKSON, J., and EUSTERMAN, G. B.: Changes in Gastric Acidity in Peptic Ulcer, Cholecystitis, and Other Diseases Analyzed with the Help of New and Accurate Technic. Arch. Int. Med., 1933, **52**, 616.

(9) BOYDEN, EDWARD A., and BIRCH, CARROLL L.: Conditions Affecting the Emptying-time of the Human Gall Bladder. Proc. Soc. Exp. Biol. and Med., 1927, **24**, 827.

(10) BIRCH, CARROLL L., and BOYDEN, EDWARD A.: Reaction of Gall Bladder to Stimulation of Gastrointestinal Tract. II. Response to Faradic Excitation of Stomach, Small Intestine, and Cecum. Am. Jour. Physiol., 1930, **92**, 301.

(11) BOYDEN, EDWARD A., and RIGLER, L. G.: A Cholecystographic and Fluoroscopic Study of the Reaction of the Human Gall Bladder to Faradic Stimulation of the Stomach and Duodenum. Anat. Rec., 1934, **59**, 427.

(12) BOYDEN, EDWARD A., and BIRCH, CARROLL L.: Reaction of Gall Bladder to Stimulation of Gastrointestinal Tract. I. Response to Substances Injected into the Duodenum. Am. Jour. Physiol., 1930, **92**, 287.

(13) BABKIN, B. P.: Nervous Control of Gastric Secretion and Effect of Vitamin Deficiency on its Production. Canad. Med. Assn. Jour., 1933, **29**, 5.

(14) BOYDEN, EDWARD A.: The Sphincter of Oddi in Man and Certain Representative Mammals. Surg., January, 1937, **1**, 25.

(15) BABKIN, B. P.: Die auessere Sekretion der Verdauungsdruesen. Springer, Berlin, 1928.

(16) IVY, A. C.: The Physiology of the Gall Bladder. Physiol. Rev., 1934, **14,** 1.
(17) WINKELSTEIN, ASHER: One Hundred and Sixty-Nine Studies in Gastric Secretion During the Night. Am. Jour. Digest. Dis. and Nutrition, 1935, **1,** 778.
(18) FATZER, HEDWIG: Ueber fraktionierte Magenausheberung nach Histamin-Verabreichung. Schweiz. med. Wchnschr., 1935, **65,** 113.
(19) KATSCH: Quoted by Berglund, *et al.*, 1935.
(20) BERGLUND, HILDUNG, JOHNSON, RICHARD, and CHANG, H. CHIEN: The Relationship between Hydrochloric Acid and Total Chlorides in Gastric Juice and on the Possibility of Standards for Gastric Secretion. Acta Med. Scand., 1935, **86,** 269.
(21) BOYDEN, EDWARD A., and RITCHIE, W. P.: The Evacuation of the Gall Bladder in Patients with Carcinoma of the Stomach. (Unpublished data.)

DISCUSSION

DR. LEO G. RIGLER (Minneapolis, Minn.): I just told Dr. Boyden that I was very glad Dr. Garland invited him here because now you will think I am a scientist also!

Of course, I am not.

I think this is a very beautiful presentation. Every time I hear it I enjoy it all the more, and the only thing I want to emphasize is Dr. Boyden's method of approach in the study of this problem.

Some of you no doubt have read some of the papers, particularly one recently presented by Kirklin and Good on the question of the demonstration of a diseased gall bladder or the apparent demonstration of a diseased gall bladder as a result of some extrinsic factor, and that type of study is extremely important in trying to rule out the errors which might occur in ordinary cholecystography.

Dr. Boyden's approach is an entirely different one in which, by using the normally visualized gall bladder, he tries to determine the effect of extra-biliary disease upon its emptying. In many ways this is a more effective method if you can do it, because approaching it from the opposite angle, the non-visualization of the gall bladder involves many other factors of error which are not present in this type of study.

I think that all of us would do well to think of these charts and to observe carefully this manner of study, because if you have seen it to any extent you realize how very accurate this procedure is, how well it works out in the study of all the physiological processes which involve the gall bladder.

DR. EDWARD A. BOYDEN (closing): I really have not anything more to say other than to answer Dr. Kirklin's question and to thank you for your very courteous attention and kind remarks.

The problem that Dr. Kirklin has mentioned is, perhaps, the most difficult of all, because in gall-bladder disease it is so hard to secure good cholecystograms and, therefore, to obtain mean curves of emptying from a large enough sample of the population. The problem could best be attacked by someone like Dr. Kirklin who has large numbers of patients coming through a clinic and who could sidetrack for special study all pathologic cases that were visualized.

THE RÔLE OF THE VEGETATIVE NERVOUS SYSTEM IN THE PRODUCTION OF MOTOR PHENOMENA OBSERVED IN THE UPPER DIGESTIVE TRACT[1]

By ALFRED C. SIEFERT, M.D., *Oakland, California*

THIS paper aims to discuss the motor phenomena which may be observed in the upper digestive tract, particularly in the stomach, and which have been observed for years by every physician interested in clinical gastro-enterological roentgenology. The writer will make an attempt to correlate these motor phenomena with facts and theories concerning the vegetative nervous system.

During the present period of morphologic roentgen diagnosis the so-called "functional findings" are looked at somewhat askance. Greater attention to them, however, is bound, in the writer's opinion, to extend the scope of our ideas, especially along the lines of the causation, and to deepen our conceptions of organic gastro-intestinal disease, and furthermore, to make us more alert to the importance of psycho-neurotic, endocrine, and allergic disturbances with reference to the digestive tract. The relationship between peptic ulcer, for instance, and psycho-neurotic influences is not a new conception. It is, in fact, observations and experiences of this phase that have aroused the writer's particular interest in the entire subject.

The observations and experiences laid down in this essay cover a period of 18 years of hospital and private roentgenologic practice. One may remark that the last years of the economic depression, especially, which have brought many individuals under heavy nervous strain, have been particularly fruitful in yielding material for the discussion in hand. Patients with gastro-intestinal symptoms directly traceable to nervous tension are found particularly among business and professional men and quite frequently among members of the medical profession.

The method of examination primarily used has been, of course, fluoroscopy, and it is frequently impossible to hold fast on roentgenograms what has been observed on the screen. Such refinements as aimed roentgenography, roentgen-cinematography, and roentgen-kymography have not been at the writer's disposal.

The motor manifestations in the upper digestive tract may be classified in three groups from which all other motor phenomena may be derived. They are: (1) tonus, (2) peristalsis, (3) sphincteric action.

Tonus.—The chief manifestation of tonus is the so-called "peristolic function, exercised by the smooth musculature of a hollow viscus. It is thus that the organ contracts closely about its contents. Tonus may be divided into three clinical states: the normal, to which we refer as the *orthotonic state;* increased contractability of a hollow viscus is called *hypertonus,* and a decrease below the normal, which allows the contents to sink by gravity to the lowest portion of the stomach, for instance, is called *hypotonus,* and, in extreme cases, *atonus.* To the tone of the longitudinal musculature of the stomach the latter owes its shape. Localized increase of tonus is termed "spasm" the "spastic incisura" and "hour-glass contraction," the latter so frequently an accompaniment of peptic ulcer, are examples. The increase in height and thickness and complexity of the gastric rugæ are expressions of increased tonus of the muscularis mucosæ of the stomach. It is rarely justifiable to interpret this condition as significant of gastritis, but a hypotonicity of the muscularis mucosæ, evidently associated with a lack of turgor in the mucosa, is quite commonly a sign of hypochlorhydria.

Peristalsis is the rhythmic contraction of the circular musculature of the stomach, beginning at the cardia on the lesser curvature, in the region where the myogenic theory of Alvarez places a pacemaker, and proceeds with increasing vigor

[1] Presented before the Radiological Society of North America, at the Twenty-second Annual Meeting at Cincinnati, Nov. 30–Dec. 4, 1936.

Fig. 1. A normal stomach, perhaps slightly hypotonic.

and depth to the pylorus. Normally, three waves travel at a time; to this, one may refer as *normal rhythm*. The time required for a given wave to reach the pylorus is greatly variable and may be called the *speed of peristalsis:* it averages about 24 seconds. Increased rhythm and speed signify motor excitement and frequently accompany organic disease, either in the stomach itself or elsewhere in the abdomen. The *obstruction* type of peristalsis is a familiar phenomenon and illustrates the fact that hypotonicity may be accompanied, at least occasionally, by increased rhythm and speed of peristaltic action.

During the act of vomiting one may now and then see antiperistalsis. One does not, however, expect to see the normal finely co-ordinated "pylorusward" peristaltic waves passing in a similar way in reverse direction.

Irregularity of peristalsis as to rhythm and speed as well as depth is occasionally seen, especially in the presence of peptic ulcer of the stomach. M. Frankel has demonstrated by roentgen-cinematographic methods a section of abolished peristalsis far beyond the confines of a peptic ulcer and its inflammatory infiltrative surroundings. He interprets this as a localized spasm of the musculature.

Chemical physical factors, actions upon the mucosa also influence the rhythm and speed of peristaltic action. The presence in the stomach of hydrochloric acid or sodium chloride acts as stimulants, while alkalis and oil have an inhibitory effect. If the opaque drink be given hot, one notes an acceleration and increased vigor of peristaltic action. When it is given cold, inhibition is noted. It is also true that psychic factors have a profound effect upon peristaltic action, as well as the tonus. This the writer has frequently observed when a patient is fearful and apprehensive of the unusual surroundings in the fluoroscopic room. Everyone has also seen peristaltic activity of the stomach begin and become increasingly vigorous on the suggestion of palatable food. Finally, blood-borne influences are not to be forgotten.

Sphincteric Action.—The opening and closing of the pylorus to an oncoming peristaltic wave is regulated probably by the so-called "duodenal-pyloric" reflex, which causes the pylorus to contract in the presence of acid chyme in the duodenum and produces relaxation when the gastric contents are neutral or alkaline. Of course, there is here, also, evidence of remote control, outside of factors which would increase the hydrochloric action of the stomach. The writer has frequently seen on examinations repeated year after year on the same patient a purely psycho-neurotic spasm of the pylorus with large six-hour retention of barium in the stomach. Similar spasm may be induced by the presence of lesions elsewhere in the gastro-intestinal tract—appendicitis, for instance—or even outside of the digestive system—kidney stone, gallstone, etc. During the act of vomiting the pyloric sphincter is spastically closed, the cardia relaxed.

Relaxations of the pylorus are seen in the presence of achlorhydria and conditions remote from the pylorus, producing achlorhydria. They are also seen in individuals presenting the hypasthenic habitus with ptotic stomach, though the peristaltic *vis a*

Fig. 2.

Fig. 3.

Fig. 2. D1730. *Diagnosis:* Ulcer of the duodenal bulb, penetrating type. Crater retains barium at six hours. The fully distended stomach showed no notable vegetative nervous system abnormalities, excepting a persistently spastic duodenal bulb. The mucosa of the stomach shows a delicate serpentine pattern; the general course of the rugæ, however, is parallel and they sweep in a gentle curve toward the pylorus. The "spidery" deformity of the duodenal bulb is considered spastic to a considerable degree. There was no six-hour residue in the stomach but a retention of barium in the ulcer crater. *Comment:* Illustrates predominantly augmentative effects in the presence of organic pathology in the duodenal bulb.

Fig. 3. D1378. Recurrent thyrotoxicosis following thyroidectomy. Present metabolic rate, plus 25 per cent. Essential hypertention. Blood pressure variable, 130/80–180/90. The clinical symptoms of the thyrotoxicosis are all quite marked. Gastro-enterostomy for duodenal ulcer in 1924: ulcer had perforated. Examination of digestive tract shows findings which one would class as "vagotonic," even in so definite a "sympatheticotonic" disease as thyrotoxicosis, and, in the writer's experience, this is quite typical. The patient exceeds the description quoted from Harvey Cushing. The mucous membrane examination is able to demonstrate a definite jejunal ulcer of the penetrating type just beyond the stoma. The duodenal bulb shows cicatricial stenosis. The gastro-enterostomy is efficient in draining the stomach. *Comments:* Illustrates in a constitutionally psychoneurotic the recurrent development of peptic ulcer, complicated by thyroid and adrenal dysfunction. The phenomena in the digestive tract are predominantly augmentative.

tergo may be insufficient to empty the stomach within the six-hour period.

The motor phenomena observed in the esophagus and in the small bowel are the same in principle as those described for the stomach.

Before taking up the vegetative innervation of the esophagus, stomach, and small bowel, it will be desirable to say a few words concerning the vegetative nervous system in general. To discuss it in detail would consume too much time, but a short discussion of the more recent ideas concerning it will undoubtedly be of interest.

The older, still widely prevailing, conception of the vegetative nervous system based principally upon the work and ideas of Gaskell, Eppinger, and Hess, and more recently of Langley and his school, is a dualistic one. Two phases, the "parasympathetic" and "sympathetic" divisions are conceived as acting in more or less strict antagonism to one another. This conception is founded not so much upon anatomical grounds or even on strictly physiologic ones, but rather upon the response to certain drugs which act on particular phases of the vegetative nervous system.

The vegetative nervous system, furthermore, is conceived as an aggregation of efferent neurons located within as well as without the central nervous system. It is, as a matter of fact, by no means as autonomic as is frequently implied or expressed, but is functionally and anatomically associated with both visceral afferent components as well as somatic afferent components. The afferent components are,

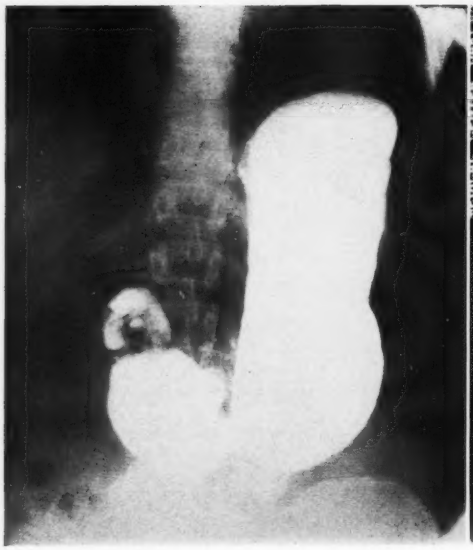


Fig. 4.

Fig. 5.

Fig. 4. D1330. *Diagnosis:* Acute duodenal ulcer. Six-hour retention of barium meal in the stomach, evidently due to spastic pylorus. Acute exacerbation of chronic appendicitis. Increased tonus of stomach, deep regular peristalsis, not increased in rate or speed; in fact, perhaps both are decreased. Pyloric antrum is perfect. Duodenal bulb shows an ulcer crater. The "halo" or "cushion" effect is due to inflammatory swelling of the surrounding mucosa which marks the ulcer as an acute one. There is considerable six-hour retention in the stomach: pressure tenderness over the duodenal bulb as well as the appendix, also muscular guard, all of which may be regarded as an overflow of afferent-sensory impulses into the cerebro-spinal and psychic sphere. *Comments:* Illustrates how peptic ulcer may be secondary to irritation of vegetative system by organic disease (infection) of the appendix. Augmentative and inhibitory phenomena are present simultaneously.

Fig. 5. D1465. *Diagnosis:* Duodenal ulcer, chronic, with organic deformity of the bulb. Attention is called particularly to the spastic incisura on the greater curvature of the stomach. The tonus of the stomach is markedly increased, and on the greater curvature the aforementioned spastic incisura is noted. At times increased rhythm of peristalsis is observed and increased speed. The pyloric antrum is persistently spastic, but may be filled out by palpatory maneuvers. An ulcer crater in the duodenal bulb can be demonstrated, with "pouching" at the base of the bulb. A large six-hour residue remains in the stomach. Arteriosclerosis of one of the branches of the celiac axis is suspected. *Comment:* Illustrates very marked predominating augmentative effects in the presence of organic disease in the duodenum of a very chronic character.

strictly speaking, not usually regarded as part of the vegetative nervous system.

From the clinical point of view, however, such a separation seems to the writer purely artificial and useful only for didactic purposes. In fact, there are anatomical and neurocytological data that have recently been brought forward by F. Kiss which would seem to show that there is a very intimate association between afferent-sensory neurons and efferent-visceral and effector-neurons.

The function of the vegetative nervous system within the view of this paper is the regulation and co-ordination of motor activities in the digestive tract. The regulation of secretory as well as circulatory activities in the digestive tract are also of great importance, though they may be studied only indirectly by the radiologist.

Functionally, in general, *augmentative* effects are ascribed to the *parasympathetic* (vagus) system, while inhibitory effects are largely considered to be the result of activity of the *sympathetic* system. It is, however, becoming increasingly difficult to explain observed facts on the basis of a strictly dualistic antagonistic theory, and many authors resort to assumptions and explanations which strike one as rather confusing. L. R. Mueller admits that the antagonism is not so constant and reliable a phenomenon as to permit the formulation of natural law, since many parts of the

gastro-intestinal tract receive augmentative as well as inhibitory impulses from either one or the other of the two vegetative divisions.

Dale and Feldberg have recently reported the formation of acetylcholine at sympathetic end-organs by stimulation of the abdominal sympathetic chain. This upsets the teaching that "sympathin," said to be identical with "adrenalin," is liberated at the end-organs of the sympathetic post-ganglionic neurons only, whereas acetylcholine has hitherto been reserved for the end-organs of the parasympathetic system.

Kuntz concludes that mutual synergism rather than antagonism of the two components of the vegetative nervous system is the mode of their functional activity. He regards the parasympathetic as bearing inhibitory as well as augmentative phases. To be sure, it is now accepted that parasympathetic and sympathetic nerves freely intercommunicate, and this may be one of the reasons for the discrepancies and paradoxes in the results of pharmacological and section experiments; also a reason for failure or lack of permanency of therapeutic results, *e.g.*, in essential hypertension on surgical intervention with the sympathetic (splanchnicotomy). A further important consideration is whether, in experiments, one is dealing with a preganglionic tract or a post-ganglionic tract.

Most interesting is the theory of Kiss which has been elaborated for the stomach by Ratkoczy in a recent communication (10). Kiss's deductions are based upon morphologic characteristics which make it possible to distinguish between the vegetative nerve cell and the afferent-sensory nerve cell. He arrives thus at the following revolutionary conclusion:

"For the assumption of the so-called cranial parasympathetic system we have no morphological basis. The cranial parasympathetic is nothing else but a part of the cranial sympathetic."

Concerning the most important representative of the parasympathetic system, *the vagus nerve*, Kiss states in substance that

Fig. 6. D1922. *Diagnosis:* Prepyloric ulcer, lesser curvature, just beyond the incisura angularis. The stomach is only moderately hypertonic. Persistent tenderness is noted on palpation over the ulcer region of the lesser curvature. The peristalsis lacks vigor; three-wave rhythm; the speed is retarded. The pylorus is, if anything, slightly relaxed. The duodenal bulb is irritable, and though well shaped, does not retain barium very well. At six hours the small residue remains in the stomach. A study of the mucosal pattern shows a definite niche on the lesser curvature. The mucosal pattern is irregular and the rugæ are increased in prominence. On the day of this examination the pylorus was quite spastic. It was possible to check this case in three months after the above examination and a more definitely formed crater is now demonstrable, with more definitely increased tonus, increased rhythm and speed of peristalsis, and spasm of the pylorus. *Comments:* Illustrates predominantly inhibitory effects in the presence of prepyloric peptic ulcer which become, as the ulcer develops, predominantly augmentative. It may be possible to draw prognostic conclusions.

"Distally to the base of the brain the vagus is no longer an unmixed (sensory nerve), but contains sensory, motor, and sympathetic fibers. The sympathetic (visceral) elements do not belong to the original vagus, but reach it by way of anastomoses with the cervical sympathetic ganglia and the spinal accessory nerve from which later it receives also its motor components.

"In man, in whom both vagi enter the abdominal cavity, the left vagus innervates the stomach with its terminal branches. The right vagus anastomoses with the celiac plexus by one or two rami, which, however, contain less than 25 per cent of the fibers originally present

Fig. 7-*A*.

Fig. 7-*B*.

Figs. 7-*A* and 7-*B*. D1915. A gross organic lesion of the digestive tract can be ruled out. There are many motor phenomena which mark this case as a disturbance of the vegetative nervous system, and, I think, on a psychoneurotic basis. To re-enumerate these symptoms, the following are of importance: The striking acceleration and irritability of the heart, the relaxation of the esophagus and aerophagia, marked hypertonicity of the stomach with increased rhythm and rate of peristalsis, moderate six-hour retention, and spastic colon. According to the referring physician, the patient is highly neurotic; in fact, psychoneurotic. The patient himself states that he is, at the present time, working under a strain and is very nervous. His chief complaints are nausea, indefinite abdominal distress, and frequent belching. Gall bladder functions normally, and shows no gross anatomical pathology. *Comment:* Illustrates predominantly augmentative effects on stomach and bowel; inhibitory on esophagus. This patient may or may not develop peptic ulcer. The sensory symptoms are considered to be produced by abnormal motor phenomena.

in the vagus, but consist mostly of sympathetic fibers.

"The effective motor and secretory innervation of the digestive tract is carried out exclusively by the sympathetic system."

According to Kiss's comparative anatomical researches and the embryologic studies of Campenhout, the celiac ganglion and the abdominal prevertebral ganglia belong to the system of the sympathetic.

The vagus, compared to the spinal nerves, shows that in its posterior sensory ganglia, the ganglion jugulare and nodosum, there are present dark-staining multipolar cells of sympathetic character in addition to the less heavily stained and larger sensory neurons. The accessory nerve represents in this analogy the motor root of a spinal nerve. To reiterate, distally to the base of the brain the vagus represents a "sensory-motor-sympathetic nerve."

Finally, concluding, Kiss says:

"In my anatomical and microscopic researches I found no morphological evidence for the assumption of a so-called 'parasympathetic system' in connection with the vagus. It appears that the parasympathetic represents only a phenomenon which may be looked upon as a negative phase of the sympathetic."

Kiss thus proclaims the unity of the vegetative nervous system in contrast to the dualistic conception, and Ratkoczy has endeavored to draw functional conclusions on this basis with reference, at least, to the stomach. The writer will quote his work further on.

The higher efferent vegetative centers, whatever conception one may accept, are located in the hypothalamus, and to distinguish here between sympathetic and para-

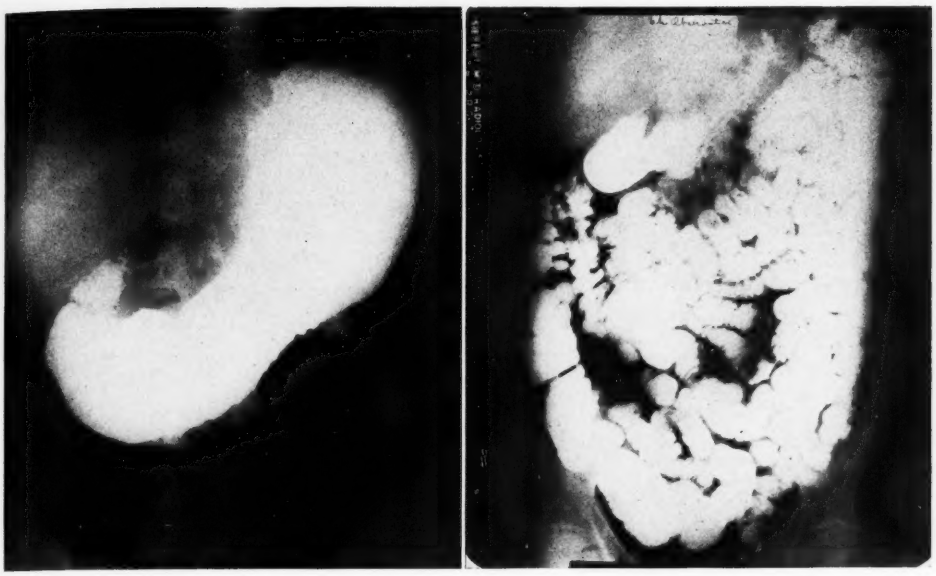


Fig. 8-*A*. Fig. 8-*B*.

Figs. 8-*A* and 8-*B*. D1792. There is no evidence of gross organic pathology in the digestive tract; even the duodenal bulb, which one might hold under suspicion, I think, one can prove innocent. Attention is, however, called to such vegetative nervous system phenomena as increased tonus of the stomach, increased tonus of the muscularis mucosæ of the stomach, increased vigor of peristalsis, and marked spasm of the pylorus. There are similar spastic phenomena in the small gut and there is a combination of hyper- and hypomotility. Through the stomach and small bowel the meal is delayed considerably beyond the normal period, whereas through the large gut the passage of the meal is accelerated. The colon shows marked spasticity. *Comment:* Illustrates in the absence of organic pathology mixed augmentative and inhibitory effects.

sympathetic centers is regarded as impossible by most authors. Vegetative centers are assumed in the cortex, but there are yet no definite anatomical proofs. From a clinical point of view, it would seem that such centers exist. Next to the hypothalamus the medulla oblongata is regarded as the site of most important vegetative centers, and lastly the spinal cord itself. Communication with the outlying paravertebral ganglia is established by the white communicating rami and, as such, the splanchnic nerves may be considered in relation to the celiac ganglion, *i.e.*, preganglionic fibers in the splanchnics. Post-ganglionic are most of the fibers proceeding from the neurons of the celiac ganglion to the organs innervated. The peripheral vagus carries largely post-ganglionic fibers.

As has already been stated, the conception of the vegetative nervous system as a system of efferent neurons, purely, is probably too narrow a conception, at any rate, to serve the clinician. That visceral sensations are more primitive and of an entirely different character from the somatic may be conceded without argument. That they exist as visceral sensations is equally certain, and they do not constitute only an overflow into the somatic-cerebrospinal sensory sphere. They may, in fact, rise in visceral afferent pathways by way of the thalamus and, especially the paleothalamus, to the cerebral cortex, and find expression in consciousness. It has been mentioned above that, according to Kiss, the sympathetic ganglia consist of both sensory and sympathetic cells. This investigator, incidentally, suggests that these ganglia may act as "transformers" in that an incoming sensory stimulus is there communicated to an efferent neuron and translated into visceral activity. Of course, reflex arcs of this nature may be instituted at higher or lower levels.

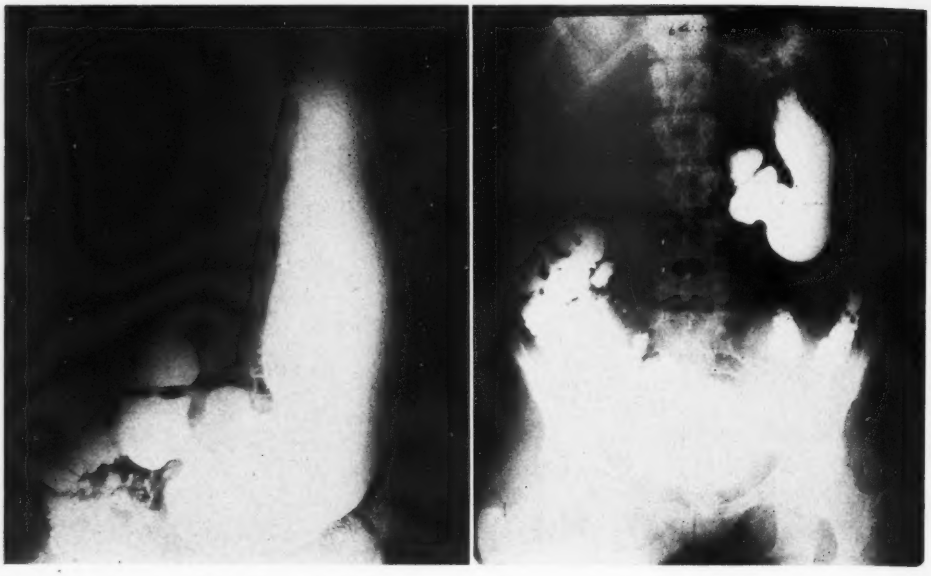


Fig. 9-*A*. Fig. 9-*B*.

Figs. 9-*A* and 9-*B*. D1912. No gross organic lesion has been found in the upper digestive tract. There are peculiar vegetative nervous system phenomena such as vigorous regurgitation of gastric contents and gastric anti-peristalsis, associated with spasm of the pylorus. There is a six-hour retention in the absence of any stenosis at the gastric outlet or any other gross organic lesion. Anti-peristalsis is noted in the duodenum. To this may be added ptosis of the stomach and bowel and spasticity of the large bowel. *Comment:* Illustrates predominantly augmentative effects, evidently from both divisions of the vegetative nervous system.

Receiving stations for afferent visceral impulses are chiefly the thalamus and paleothalamus, which are regarded as the centers of "primitive sensation" and of "awareness," and stand in close relationship to the center of efferent visceral impulses, the hypothalamus, as well as the cortical sensory-motor, and visceral-efferent centers.

Concerning the innervation of the esophagus, stomach, and bowel we may make a brief statement: The esophagus is innervated by the vagus, the right and left forming a plexus about it. Sympathetic fibers come by way of the cervical sympathetic and celiac plexus to the esophagus.

The stomach is innervated by the vagus, the left one innervating the anterior and cardiac portion, the right one the posterior and pyloric portions. The sympathetic innervation is derived from the celiac plexus which, according to different authors, may be either purely sympathetic or mixed. The same is generally true of the small bowel. In the stomach the "autonomic" system is regarded as acting by mediation of the myenteric neurons, presently to be discussed.

The vegetative system is an "extrinsic" system and is accorded only regulatory rather than initiatory function. Speaking for the stomach, there exists, in addition to the extrinsic, an "intrinsic" nervous system, which some have called the "enteric," "myenteric" or "intramural" system. The plexus of Auerbach is located between the longitudinal and the circular muscular coats of the stomach, that of Meissner below the mucosa. The conception of the majority of writers is that it consists of true neurons, effectors and sensory in nature, which form complex connections with one another and are able to act reflexly to irritation and stimuli conveyed by afferent or sensory neurons located intramurally. To be sure, some authors, like Bayliss and Starling and more recently Alvarez, have emphasized the automatism of smooth muscle which

Fig. 10. D2001. No gross organic disease has been demonstrated in this patient's digestive tract. The peculiar regurgitation of the gastric contents into the esophagus, and loud belching, especially when pressure is made on the pyloric portion of the stomach, are, I think, vegetative neurotic phenomena. In addition to this, there is a considerable residue in the stomach and duodenum at six hours, yet it is not possible to demonstrate an obstructive lesion of any kind. The cecum shows non-rotation. *Comment:* Figures 7 to 10, inclusive, may be said to illustrate the "nervous type" in which peptic ulcer is likely to develop. We should learn how to treat peptic ulcer preventively.

Fig. 11. D1949. *Diagnosis:* Appendicitis, chronic, with recent acute exacerbation (surgical diagnosis following gastro-intestinal examination). The patient has had repeated attacks of nausea and vomiting, associated with more or less severe pain in the lower right abdominal quadrant. The highlights of the examination are the following: ptosis of the stomach with hypertonus and hypertonicity of the muscularis mucosæ, peristalsis not increased in rhythm or speed, pylorus spastic; in fact, so much so that good filling of the duodenal bulb is only occasionally obtained by manipulation under the fluoroscope. At six hours there is considerable residue in the stomach. The cecum is fixed, and distinctly tender mesially. At 24 hours the appendix may be visualized, and there is sharply localized tenderness elicited on pressure over it. The roentgen diagnosis: appendicitis, chronic, with present acute or sub-acute exacerbation. The phenomena observed in the stomach are reflex in nature, due to irritation of the vegetative nervous system, and quite common.

rhythmically contracts and in a purposeful direction—gradient—after having been deprived of all extrinsic innervations. The intramural plexuses are also regarded by some as a syncytium, the purpose of which is conduction, thus entirely eliminating the neuron doctrine.

Pharmacologically, pilocarpine, physostigmine, and choline stimulate the parasympathetic; atropine and its allies paralyze the parasympathetic. Adrenaline is said to stimulate the sympathetic division; also ephedrine, benzedrine, and cocaine. Ergotamine and ergotoxine depress the sympathetic division. Nicotine, according to Langley, interrupts the synaptic connection between the pre-ganglionic and post-ganglionic neurons in each division of the autonomic system. The study of drug action on the nervous system of the digestive tract, with diagnostic or therapeutic employment in view, should be a part of our roentgenological program.

Now, to quote from Ratkoczy, who utilizes the experiments of Bickel and Watanabe as well as his own roentgenological observations:

"If a dog be injected with acetylcholine, marked increase of the gastric tonus is observed. Peristalsis stops and the emptying time is prolonged.

"If pilocarpine be injected, hypertonus of the stomach, increased peristalsis, and shortening of the emptying time is the result.

"Adrenaline, when injected into a dog, shows

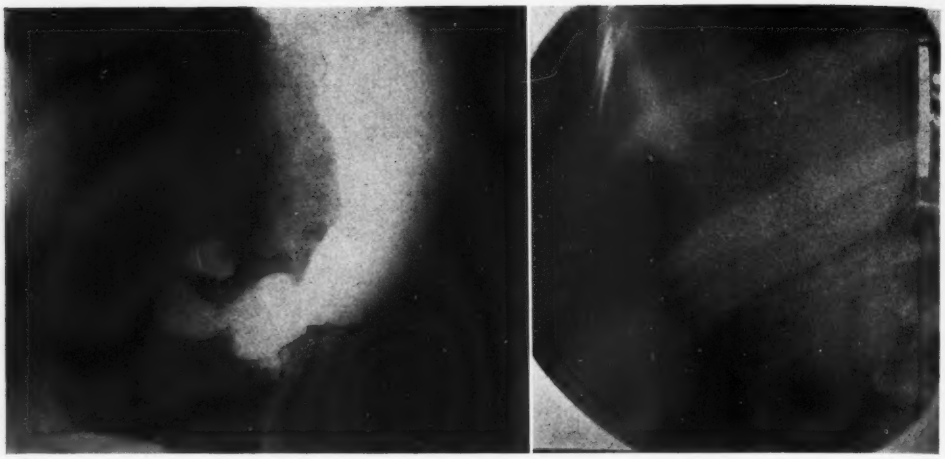


Fig. 12-*A*. Fig. 12-*B*.

Figs. 12-*A* and 12-*B*. C892. *Diagnosis:* Cholelithiasis, with non-functioning gall bladder according to the Graham test. The stomach, aside from well-marked hypertonicity and moderate spasm of the pylorus, does not show any reflex vegetative phenomena. *Comments:* Figures 11, 12-*A*, and 12-*B* illustrate how gastric motor-effects may be produced by afferent impulses rising from disease elsewhere in the abdomen. The efferent impulses are usually augmentative in character, depending on the strength of the afferent impulses.

two phases of reaction: first, tonic contraction, irregularity of peristaltic action, even to antiperistalsis and vomiting; the second phase follows after a few minutes and is characterized by marked hypotonus, abolition of peristalsis, and marked delay in emptying.

"Atropine produces atony, abolition of peristalsis, and increased emptying time."[2]

In explanation of these experiments, Ratkoczy says that they are much more readily explained by the assumption of unity or synergism of the vegetative system and more logically than by the dualistic antagonistic theory. "It is," he says, "much more logical to presume that the pylocarpine group exerts a stimulus on the entire vegetative nervous system, whereby all motor activity is increased, while atropine and its allies paralyze the entire system. Adrenaline has a two-fold action; an excitory one, followed by a paralyzing one."

To quote further:

(1) "Intrathoracic double vagotomy immediately above the diaphragm produces hypotonicity and decreased peristaltic action as well as increase of emptying time. These abnormalities are compensated, after a lapse of time, to a certain degree.

[2] This quotation has not been translated *verbatim*, but conveys the meaning.

(2) "Intrathoracic double sympathico-splanchnicotomy immediately above the diaphragm produces hypertonicity and increase of peristalsis with decrease of the emptying time. After a lapse of time these phenomena subside and leave the stomach slightly hypotonic.

(3) "Extirpation of the celiac ganglion produces an immediate and marked hypertonicity and hyperperistalsis which seems to exceed even the phenomena following sympathetico-splanchnicotomy.

(4) "After combined double vagotomy and sympathetico-splanchnicotomy the stomach is markedly hypotonic, peristaltic activity is markedly decreased and the emptying time is considerably prolonged.

A compensation of these disturbances is brought about with more difficulty than if only one division has been cut."

In discussion the author says:

"According to our conception mostly postganglionic fibers are cut with the vagus, the function of which is an augmentative one. The result is naturally a decrease of all motor manifestations until other nerves with similar augmentative action (*i.e.*, the sympathetic) take over the function of the cut pathways.

"To explain the results of experiments Nos. 2 and 3 it is necessary to review the anatomical construction of the splanchnic. As has been mentioned, the splanchnic contains mostly preganglionic pathways which may be compared, according to their anatomical level, to the cortico-muscular tracts. The section of the

former, accordingly, produces similar consequences as does a lesion of the pyramidal tracts in the somatic motor sphere. [This, of course, merely in analogy.] Since the pre-ganglionic pathways contain not only central augmentative impulses, but also central inhibitory impulses, the consequence of their section is increased motor activity on part of the intramural neurons excited by hematogenous stimuli, or they are stimulated by intramural reflexes. This hyperfunction, however, is not of long duration. The intramural ganglia cannot do without the central augmentative impulses, the pathways of which have been cut in sectioning the splanchnic. Thus the end-result of splanchnic section is the same of that of vagus section, namely, hypotonus, decrease of muscular activity. According to our conception, it is self-evident that the elimination of afferent pathways must finally result in hypofunction.

"The result of experiment No. 4 cannot at all be explained on the antagonistic theory. The elimination of two antagonistic pathways should result in compensation.

"Splanchnicotomy or the extirpation of the celiac ganglion should compensate for the atonic effect of vagus section. Instead of that we see hypotonicity, hypoperistalsis, increase of emptying time reaching most extreme degrees. The result of the experiment, according to our conception, proves that the elimination of two augmentative tracts must result in a decrease of muscule activity, which is not balanced by simultaneous elimination of the inhibitory pathways running in the splanchnics."

Combination experiments, according to Bickel, quoted by Ratkoczy, are the following:

(5) "Pilocarpine increases the peristaltic activity of the stomach excessively after the splanchnic has been cut.

(6) "Pilocarpine produces nearly the same reaction when the vagus has been sectioned as it does with the normally innervated stomach.

(7) "After the vagus and splanchnic both have been sectioned, the action of the pilocarpine is notably weaker.

(8) "If the vagus has been sectioned and the celiac ganglion extirpated, the action of pilocarpone is still weaker."

Comments on Experiment No. 5.—"The explanation is the same as in experiments Nos. 2 and 3, *i.e.*, analogous to a lesion of the pyramids."

Comments on Experiment No. 6.—"If the vagus (parasympathetic) carried only augmentative phases, then the elimination of these would necessarily be evident in a much greater measure. This experiment corroborates our opinion that the vagus is only a very small part of the augmentative system, the loss of which may be comparatively readily compensated by the other parts of the system, namely, the sympathetic."

Fig. 13. D1819. *Diagnosis:* Digestive disturbances on allergic basis. Patient, aged 10 years, complains of pain in epigastrium immediately following breakfast, together with nausea and anorexia. Within from fifteen minutes to half an hour these symptoms disappear and the child feels perfectly well, but refuses further food. At the noonday and evening meals these symptoms are usually not observed. Roentgen examination reveals regurgitation of opaque drink which is regarded as a psychical phenomena indicating patient's dislike of the barium mixture. Subsequently, stomach fills readily and smoothly; its tonus is normal; peristalsis is regular and vigorous, not increased in rhythm or speed. The pylorus is not spastic; on the contrary, somewhat relaxed. The duodenal bulb fills well and smoothly. The descending duodenum shows no abnormality in appearance and mucosal structure. One notes, however, persistent anti-peristalsis; after about fifteen minutes to half an hour this ceases and the patient no longer complains of pain. The stomach empties well within the six-hour period.

Comments on Experiments Nos. 8 and 9.—The results of these experiments are equally classical proofs of the unity of the vegetative system as is that of No. 4. The splanchnic system, accordingly, cannot be looked upon as a simple inhibitory system.

"It is also difficult to conceive that the relatively thin peripheral vagus should be destined for the purpose of transmitting only augmentative impulses and that a system as extensive as that of the splanchnic should have a solely inhibitory function."

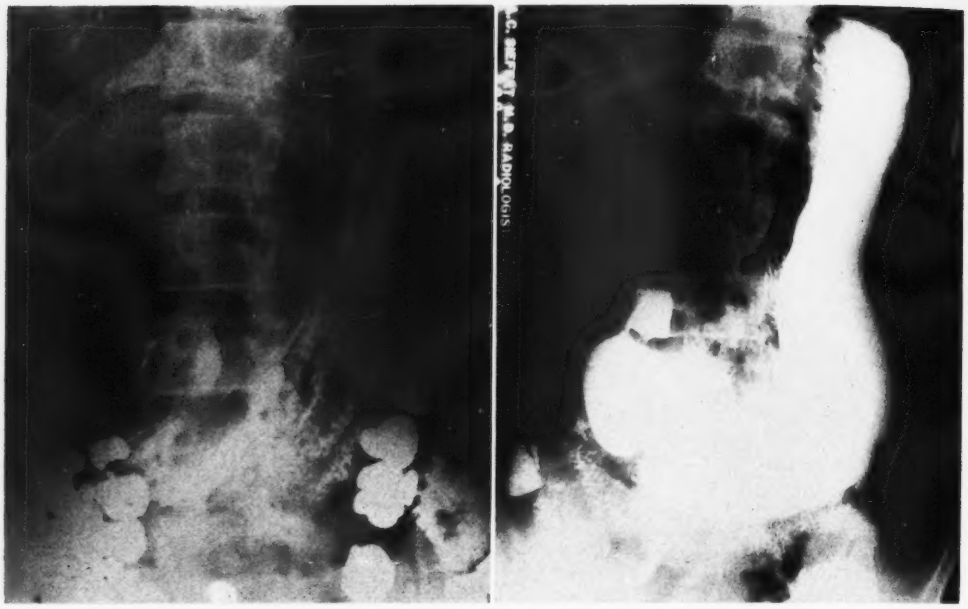


Fig. 14-*A*. Fig. 14-*B*.

Figs. 14-*A* and 14-*B*. D2000. No gross organic pathology; important disturbances of the vegetative nervous system. These findings are: well marked ptosis of the stomach and bowel; hypotonicity of the stomach; lack of vigor and decreased rhythm and speed of gastric peristalsis. There is a six-hour retention in the stomach. A gastric analysis was requested on the basis of the appearance of the gastric mucosæ; achlorhydria was reported. *Comment:* Illustrates the effects of debilitating general disease as well as heriditary habitus.

The author concludes that there exists only a single division of the vegetative nervous system, and drops the entire antagonistic theory, together with the parasympathetic. He states that he has been able to show that all motor phenomena in the stomach may be thus explained without having to resort to exceptions or paradox reactions or even reversed reactions.

I have quoted the work of Kiss and Ratkoczy in greater length, not because I accept their theories without further criticism as the only possible explanation of motor phenomena observed in the upper digestive tract, but rather because they are novel and tend to lead away from a multitude of contradictions and exceptions, provided their premises are true. It follows, too, that with such great diversity of opinion, it is possible only to register observations, until a satisfactory theoretical explanation is forthcoming.

As an introduction to the illustrations the writer would like to discuss for a moment visceral conditions which are felt as pain. They are primarily: (1) distention, (2) contraction or spasm, (3) pain resulting from irritation of the parietal peritoneum. The latter are somatic and not primarily visceral in character. Such sensation as nausea, anorexia, burning pain, and hunger pain, as shown by Carlson, are dependent upon *motor* abnormalities of the esophagus, stomach, and the upper small intestine. These phenomena may also result from conditions higher in the vegetative nervous system, such as sea- or air-sickness, for instance, which has to do with the organ of equilibration. To emphasize: the visceral motor phenomena under discussion may be due to afferent sensory effects, *i.e.*, visceral pain.

Co-ordinated vomiting is possible only if both divisions of the vegative system are intact. Thus, the splanchnic augmenta-

tive impulses close the pylorus, and the inhibitory impulses, travelling in the vagus, relax the cardia and the cardiac opening. Augmentative impulses travelling in the vagus, produce the contraction of the stomach and the antiperistalic effort necessary for elimination of the gastric contents. With ingested toxic substances, it is easy to see how intramural reflexes may start the mechanism.

Concerning gastro-intestinal allergy, the writer wishes to state that, having seen a number of such cases corroborated by allergic manifestations in the skin, he considers allergy a vegetative nervous system phenomenon, in that certain substances, foods, etc., are toxic to the vegetative system in some individuals at some time, and elicit more or less characteristic motor responses.

In the discussion of gastric ulcer the writer has alluded in the introduction to his own experiences as to a neurogenic factor in the causation of peptic ulcer. Many authors corroborate this opinion and have adduced a certain amount of proof for their contention; notably, Harvey Cushing, from whose essay on "Peptic Ulcer and the Interbrain," I wish to quote only one short paragraph.

"All clinicians are familiar with the facts that highly strung persons are particularly susceptible to nervous indigestion and associated ulcer. The ulcers become symptomatically quiescent or even tend to heal when patients are mentally and physically at rest; at that, symptoms are prone to recur as soon as the victims of the disorder resume their former tasks and responsibilities. Though this emotion of psychic aspect of the ulcer problem has been frequently emphasized in the past, the locus of primitive emotions and their relation to parasympathetic discharge and vagotonia has only come to be partly understood in recent years. It is proper, therefore, at the outset to disclaim any pretence toward a novel explanation of the pathogenesis of ulcer. At the same time, the hope is expressed that what will be forthcoming will serve in a measure to reconcile the several conflicting hypotheses, many, if not all of which will doubtless contain certain elements of truth."

Fig. 15. D1721. *Diagnosis (roentgen)*: Chronic appendicitis with sub-acute exacerbation. Noteworthy are the high position and "steer-horn" formation of the stomach, marked hypertonus, increased rhythm and speed of peristalsis, spastic antrum, hypertrophy of pylorus. Normal emptying time. The motor phenomena in the stomach are probably reflex to the sub-acute exacerbation of a chronic appendicitis. The important feature is the hypertrophy of the pylorus which the writer lays to persistent spasm as an etiologic factor. *Comment:* Illustrates organic changes brought about by long-continued vegetative nervous disturbances.

SUMMARY AND CONCLUSION

(1) A sketch is presented of normal and abnormal motor phenomena, roentgenologically observable in the upper digestive tract, together with some of the modern ideas and conceptions of the vegetative nervous system in its rôle of regulating and initiating such motor phenomena.

(2) Attention is directed to the idea that the vegetative nervous system plays an important part in the causation, not only of functional disturbances, but also of organic disease, particularly, of peptic ulcer.

(3) Attention is also called to the causation of digestive disturbances through the mediation of the vegetative nervous system in endocrine and allergic disturbances, and last but not least, in psychoneurotic abnormalities.

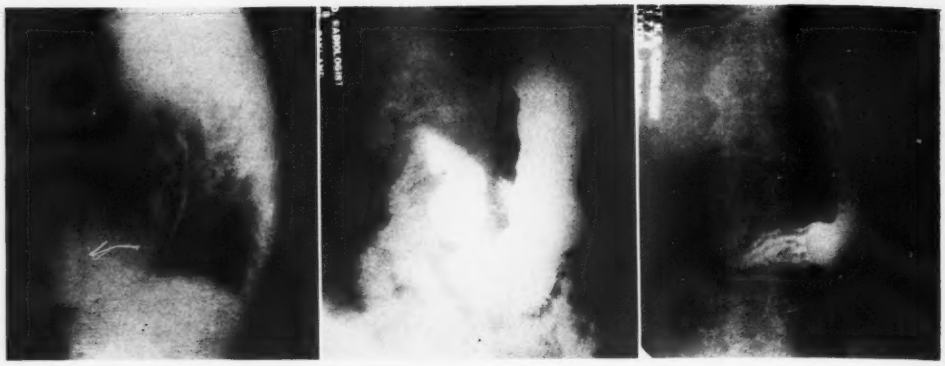


Fig. 16-*A*. Fig. 16-*B*. Fig. 16-*C*.

Figs. 16-*A*, 16-*B*, and 16-*C*. D1775. *Diagnosis:* Thyrotoxicosis. Metabolic rate, plus 34 per cent; clinical symptoms of thyrotoxicosis are marked; blood pressure, 210/80. Digestive symptoms: lack of appetite, nausea, and frequent diarrhea. The roentgen findings in the gastro-intestinal tract are typical of so-called "vagotonia," but it is more probable that the augmentative impulses of the sympathetic system are stimulated over the inhibitory, in case of the digestive tract. Noteworthy are the hypertonus of the esophagus with definite peristaltic action, practically no aerophagia, hypertonicity of the stomach, gastric peristalsis increased both in rhythm and in speed. Spastic pylorus; anti-peristalsis in the descending duodenum; hypertonicity of the small gut; hypertonicity of the muscularis mucosa of the stomach. Patient complains of anorexia and inability to swallow, the latter recognized as imaginary; also a feeling of fullness and pain referred to the stomach. *Comment:* Illustrates typical endocrine effects on the vegetative nervous system of the digestive system, both as to afferent and efferent phenomena.

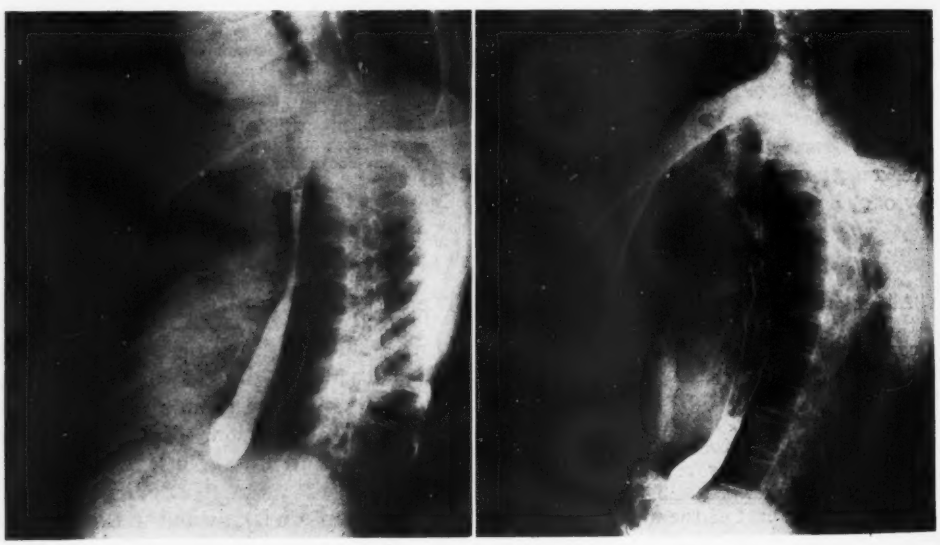


Fig. 17. Fig. 18.

Fig. 17. D1793. *Diagnosis:* Normal esophagus.

Fig. 18. D1983. *Diagnosis:* Relaxation of esophagus, with slight spasm at cardia. Note marked aerophagia. Ptosis of stomach and bowel.

(4) With these observations in mind, it is not only possible but actually incumbent upon the physician to practise preventive therapy of peptic ulcer and other gastro-intestinal disturbances.

(5) From a scientific point of view, the old clinical pictures of "vagotonia" and "sympatheticotonia" should be discarded, as they are no longer tenable. The writer suggests the expressions "predominantly

Fig. 19-*A*.

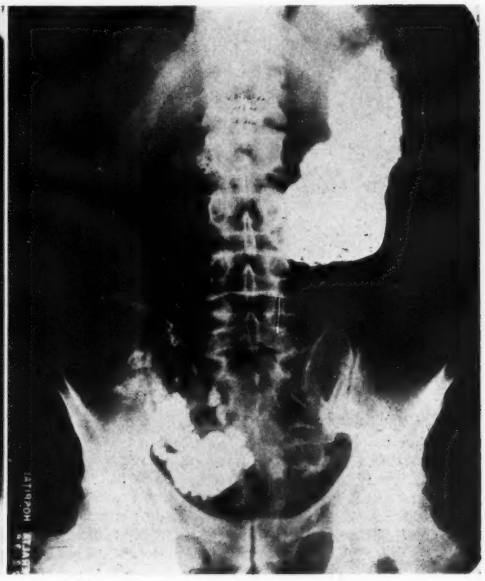


Fig. 19-*B*.

Figs. 19-*A* and 19-*B*. (Dr. Coat.) *Diagnosis:* obstruction at gastric outlet. Ulcer niche visible in oblique projection of duodenal bulb. At six and 24 hours a large residue is seen in the stomach. Effect of tincture belladonna (atropine); there is now, on attaining the physiological limit, a relatively moderate decrease in tonus and in vigor, rhythm, and speed of peristalsis. There is definite relaxation of the pylorus, however, so that it is now possible to demonstrate the ulcer crater *en face* in the sagittal projection. (See Figs. 19-*C* and 19-*D*.)

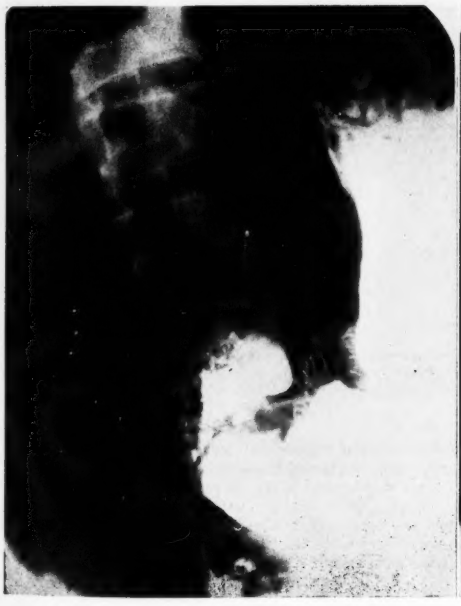


Fig. 19-*C*.

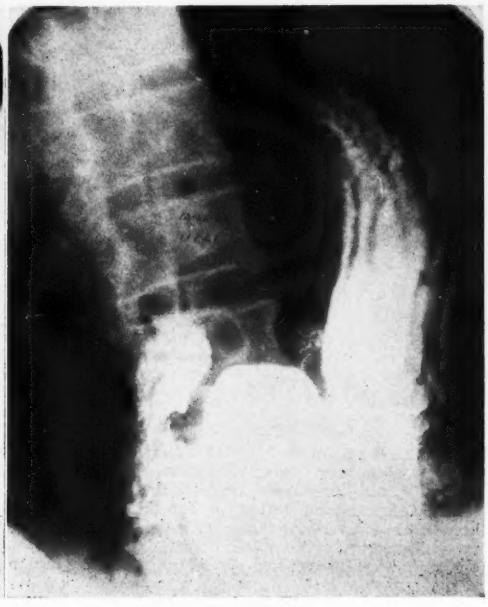


Fig. 19-*D*.

Figs. 19-*C* and 19-*D*. See caption under Figs. 19-*A* and 19-*B*.

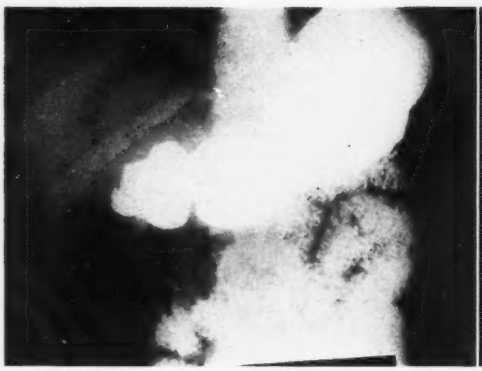


Fig. 20-*A*.

Fig. 20-*B*.

Figs. 20-*A* and 20-*B*. D1957. *Diagnosis:* Duodenal ulcer. Pain in the epigastrium about two hours after meal, typical hunger pain. The pain is, however, not a daily occurrence and the patient may go several weeks without it. The stomach is high and transverse. Tonus markedly increased. Peristalsis is regular, not increased in rhythm or speed. Pylorus is spastic. It is difficult to visualize the duodenal bulb. No six-hour retention. On the following day 1 mmg. of benzedrine sulphate was given and after half an hour an opaque drink was administered. The pyloric sphincter was found to be definitely relaxed and, peculiarly, the tonus and the peristaltic activity definitely increased. The film is shown to demonstrate this action of benze drine. The duodenal ulcer was demonstrated subsequently by means of showing the mucosal pattern.

Fig. 21-*A*.

Fig. 21-*B*.

Figs. 21-*A* and 21-*B*. D1790. *Diagnosis:* Organic obstruction of gastric outlet, probably by a duodenal ulcer or ulcer scar. Noteworthy are marked hypertonus, deep vigorous peristalsis, which, while not increased in rate, impresses one as obstruction peristalsis, since the stomach occasionally dilates and there is absence of peristaltic activity. The duodenal bulb may be seen but it is very small and distorted. At six hours there is a very large residue in the stomach; at 24 hours the stomach is empty. Benzedrine sulphate, 30 mmg., was given and one-half hour later the stomach was re-examined. There is no relaxation of the gastric outlet, but again there is increased tonus and increase in peristaltic activity.

Comments on Figs. 19-A to 21-B, inclusive. These illustrate the effects of two drugs used commonly in roentgen diagnosis. Of the two, in the writer's opinion, benzedrine is the more satisfactory, although there is the disadvantage that it is unsafe to use in cardio-vascular disease, particularly hypertension.

The relaxing effects of atropine, in the author's experience, are not so satisfactory, at least not with moderate dosage. The writer has observed absolutely no effects on a spastic colon, for instance, with long-continued administration of atropine and its allies, particularly hyoscine.

augmentative," or "predominantly inhibitory" for the phenomena observed.

(6) The study of drugs acting directly upon the vegetative nervous system with diagnostic as well as therapeutic aims is incumbent upon the modern roentgenologist.

(7) Concerning the illustrations offered in this paper, it is to be emphasized that they are meant as types, and are to convey the idea of motor phenomena rather than to demonstrate pictures of organic pathology, which they may or may not show.

REFERENCES

(1) KUNTZ, ALBERT: The Autonomic Nervous System. Lea & Febiger, 1934.

(2) MÜLLER, L. R.: Lebensnerven und Lebenstriebe. Springer, 1931.

(3) JELLIFFE, S. E., and WHITE, W. A.: Diseases of the Nervous System. Lea & Febiger, 1935.

(4) CUSHING, HARVEY: Papers Relating to the Pituitary Body, Hypothalamus, and Parasympathetic Nervous System. Charles C. Thomas, 1932.

(5) KISS, F.: Anatomisch-histologische Untersuchungen ueber das sympathisch Nervensystem. 1932.

(6) LIVINGSTON, W. K.: The Clinical Aspects of Visceral Neurology, with Special Reference to the Surgery of the Sympathetic Nervous System. Charles C. Thomas, 1935.

(7) EUSTERMAN, GEORGE B., and BALFOUR, DONALD C.: The Stomach and Duodenum. W. B. Saunders Co., 1936.

(8) ALVAREZ, WALTER C.: The Mechanics of the Digestive Tract. Paul B. Hoeber, Inc., 1928, third ed.

(9) FORSSELL, GÖSTA: Über die Beziehung der Röntgenbilder des menschlichen Magens zu seinem anatomischen Bau. Fortschr. a. d. Geb. d. Röntgentherapie (Ergänzungsband), 1913, **30.**

(10) RATKOCZY, N.: Neue Grundsteine zur funktionellen Röntgendiagnostik des Magens. Fortschr. a. d. Geb. d. Röntgenstrahlen, March, 1936, **53.**

(11) ASSMANN, HERBERT: Die klinische Röntgendiagnostik der inneren Erkrankungen, Vol. 2. Vogel, 1934.

(12) ZOLLINGER, R.: Significance of Pain and Vomiting in Cholelithiasis. Jour. Am. Med. Assn., Nov. 23, 1935, **105,** 1647–1652.

(13) MYERSON, A., and RITVO, MAX: Benzedrine Sulfate and Its Value in Spasm of the Gastro-intestinal Tract. Jour. Am. Med. Assn., July 4, 1936, **107,** 24–26.

(14) DOJUT, BENJAMIN, and STEIN, EPHRAIM: Gastro-ileostomy and Gastroileac Ulcer. Am. Jour. Surg., August, 1936, **33** (N.S.), 263.

(15) GOLDEN, ROSS: Observations on Small Intestinal Physiology in Presence of Calcified Mesenteric Lymph Nodes. Am. Jour. Roentgenol. and Rad. Ther., March, 1936, **35,** 316–323.

(16) MITCHELL, EDWARD C., and SEMMES, R. E.: Hirschsprung's Disease: Its Pathological Physiology and Apparent Cure of Two Cases under Observation for Two and Three Years Following Sympathectomy. South. Med. Jour., July, 1933, **26,** 606–614. Abstracted in Year Book of Radiology, 1934, p. 217.

(17) KIRKLIN, B. R.: Duodenitis and its Roentgenologic Characteristics. Year Book of Radiology, 1934, p. 205.

(18) PENDERGRASS, EUGENE P., and COMROE, B. I.: Roentgen Study of the Gastro-intestinal Tract in Chronic Idiopathic Adult Tetany. Am. Jour. Roentgenol. and Rad. Ther., May, 1935, **33,** 647–656.

(19) STUMPF, PLEIKART: Movements of Mucous Membrane Folds of the Stomach with Flat Kymograph. Fortschr. a. d. Geb. d. Röntgenstrahlen, March, 1936, **53,** 356–363. Abstracted in Year Book of Radiology, 1936, p. 241.

(20) RAVDIN, I. S., PENDERGRASS, E. P., JOHNSTON, C. G., and HODES, P. J.: Effect of Foodstuffs on Emptying of Normal and Operated Stomach and Small Intestinal Pattern. Am. Jour. Roentgenol. and Rad. Ther., March, 1936, **35,** 306–315.

(21) ABBOTT, W. O., and PENDERGRASS, E. P.: Intubation Studies of Human Small Intestine: Motor Effects of Single Clinical Doses of Morphine Sulphate in Normal Subjects. Am. Jour. Roentgenol. and Rad. Ther., March, 1936, **35,** 289–299.

(22) BERG, H. H.: Röntgenuntersuchungen am Innenrelief des Verdauungskanals. Thieme, Leipzig, 1930.

(23) CHAOUL, HENRI, and ADAM, ALBERT: Die Schleimhaut des Verdauungskanals im Röntgenbild. Urban & Schwarzenberg, 1931.

DISCUSSION

DR. B. R. KIRKLIN (Rochester, Minn.): The subject of Dr. Siefert's paper is broadly stated and might involve a host of problems in the physiology and pathology of the upper portion of the alimentary canal. One important phase of the theme would be the probable influence of the sympathetic nervous system in the production of gastrospasm, and in that phase I am keenly interested.

Almost since the beginning of gastro-intestinal radiology, two varieties of gastrospasm have been recognized—one produced by lesions of the stomach and conveniently designated "intrinsic spasm"; the other associated with extra-gastric diseases or conditions and commonly called "extrinsic spasm."

Examples of intrinsic spasm include the rarely seen incisura or constriction in the plane of an ulcer, the curling of the antrum that often accompanies benign ulcer of the distal lesser curvature, and the spastic contraction and distortion of the prepyloric region that is usually associated with ulcers near the pylorus.

Spasm from extrinsic causes also assumes a variety of forms, including general increase of muscular tone, spasticity of the pylorus, transitory hour-glass contraction, travelling incisuras, crenulation of the greater curvature, antral distortion, and gross deformities of the stomach.

Because spasm from extrinsic sources resembles that caused by gastric disease, interferes with inspection of the mucosal relief, and hampers filling and visualization of the duodenum, it has always received solicitous attention, and various measures—including the administration of antispasmodics—have been employed to prevent or remove this handicap.

Through all the years it has been assumed that the chief causes of extrinsic gastrospasm are duodenal ulcer, disease of the gall bladder, and chronic appendicitis, and that other intra-abdominal affections occasionally are responsible. That such diseases may reflexly, through the autonomic nerves, give rise to spasm cannot be denied for the association has been observed repeatedly. Besides, the stomach is notoriously sensitive to pathologic conditions elsewhere and many diseases that do not implicate the stomach organically are attended with gastric symptoms, some of which are logically attributable to reflex spasm.

But of late I have become convinced that the most common cause of the spastic manifestations so often encountered at roentgenoscopic examination is not disease of some organ other than the stomach but certain emotions of the patient, particularly fear and anger. This belief is in consonance with the accepted fact that emotional states may markedly affect gastric tone, secretion, and motility.

At all events, when the patient under roentgenoscopic examination is apprehensive or vexed, he assumes a defensive attitude. His abdominal muscles are tensed and the resulting increase of intra-abdominal pressure pushes the stomach upward and deforms it. Frequently such deformities have been mistakenly attributed to gastrospasm, but after the patient has been persuaded to relax his abdominal muscles, really spastic manifestations are often evident. Gastric tone is abnormally increased, the stomach is drawn up under the ribs and sometimes distorted, and the pylorus remains obstinately closed. Consequently, the necessary manipulations of the stomach cannot be carried out and it is difficult or impossible to exhibit the duodenal bulb.

Even slight degrees of vexation or alarm on the part of the patient may be sufficient to induce such spasm. For example, I recently had a patient—a woman—whose stomach was so spastic that I could not examine her. On inquiry I learned that my assistant had grasped her hand abruptly when she entered the room and led her to the screen apparatus instead of merely directing her where to stand. Her resentment at this seeming indignity was responsible for her intractability.

Again, a few days ago I examined a man without difficulty but asked for a re-examination to confirm the findings. Next day when he appeared for the second examination, his stomach was spastic and unmanageable. When he was questioned, he admitted that the request for re-examination had alarmed him because he regarded it as an indication that he had a grave affection of the stomach.

I have found, also, that when I am irritable my patients acquire my mood and become recalcitrant!

So certain am I that emotional stress induces gastrospasm that in my screen room procedure many precautions are taken to avoid vexing or alarming patients. Attendants are required to speak in low and pleasant tones, to shun any appearance of haste or excitement, to keep their hands off patients, and to treat them with all respect and dignity. During the examination I try with brief but friendly conversation to gain the patient's confidence and promote his serenity.

By these measures my work has been made easier, re-examinations have diminished, and accuracy in diagnosis has been approached more closely.

NON-CARCINOMATOUS TUMORS OF THE STOMACH

By RAY A. CARTER, M.D., *and* DONALD R. LAING, M.D., *Los Angeles, Calif.*

From the Department of Radiology, Los Angeles County Hospital

THE relatively infrequent non-carcinomatous gastric tumors vary widely in gross morphology and associated roentgen manifestations. Benign tumors are frequently recognized (4); the others are seldom diagnosed roentgenographically. When a carcinoma or a benign tumor is simulated, the confusion is not so serious, if all gastric tumors are considered at least potentially malignant, and, when possible, subjected to microscopic proof. However, under a diagnosis of polyposis, hypertrophic gastritis, benign ulcer, or syphilis, surgery or irradiation may be withheld. Also sarcoma, still resectable, may resemble frankly inoperable gastric carcinoma or abdominal tumor (9).

It may then be justifiable, in spite of the generally ackowledged absence of reliable roentgen signs, to survey these variegated lesions together for any predilections, roentgenographic or clinical, which will justify suspicion of their presence.

Benign tumors of the stomach have been considered rare, but recent literature has established that they are not as uncommon as previously supposed. Careful roentgenologic and pathologic studies have lifted these lesions from comparative obscurity.

The incidence of benign tumors has been found to be 1.3 per cent by Eusterman and Senty (14), less than 2 per cent by Kirklin and Weber (quoted by Rigler and Ericksen, 32), about 4.5 per cent by Lockwood (24), and recently Rigler and Ericksen (32) put the figure at approximately 11 per cent. Reiniets (31) made a careful examination in 200 consecutive postmortems and found small single or multiple gastric leiomyomas in 17 per cent, demonstrating that a meticulous search raised the pathologic incidence considerably.

Benign tumors had a predilection for the pylorus in 69 per cent of the cases, 25 per cent being situated in the body and 5 per cent in the cardia. The Balfour series (2) of 58 cases of benign tumor of the stomach is probably a fair representation of predilective sites. Of 14 single adenomatous polyps, one was on the anterior wall, four on the posterior wall in the body, and nine on the posterior wall in the prepyloric region. In four cases of adenoma, three were near the pylorus and one on the anterior wall near the mid-stomach. Under the general group of fibromas, myomas, fibromyomas, adenomyomas, and myxofibromas, 10 tumors were situated near the pylorus, two in the antrum, two in the midgastric region, and one in the cardia.

In our case of gastric tuberculoma the lesion was on the greater curvature in the prepyloric region (Fig. 1).

Overfilling of the stomach with the barium was the commonest cause of the failure to make a diagnosis. The rugal pattern was obliterated and often a tumor of relatively moderate size was hidden. Adequate manipulation and palpation, pressure on the stomach, and films made during roentgenoscopy gave better visualization and localization. Small defects of the anterior and posterior walls of the stomach were brought out by films of heavy penetration (32).

The vague and protean symptoms of these tumors have been the cause of the dismissal of many patients as being neurotic or having a gastric disturbance of a reflex nature. However, recent data have shown us that there are certain signs and symptoms which are at least strongly suggestive of benign gastric tumor. The manifestations are dependent upon the size and location of the tumor and anemia or ulceration. Pedunculation of the tumor

Fig. 1.

Fig. 1. Tuberculoma, with sharply localized, smooth, rounded encroachment upon the gastric lumen. Resembles benign tumor or endogastric sarcoma. There was far advanced, pulmonary tuberculosis.

has been found responsible for intussusception into the duodenum, strangulation, the syndrome of intermittent gastric ileus of the mechanical type (26). Of the group 10 per cent had pyloric obstruction (14).

Gastro-intestinal hemorrhage and anemia may be the only and the most important signs and symptoms in many cases (14). Many patients had some form of dyspepsia. Judd and Hoerner (20) found this to be true in 60 per cent of their cases; others simulated peptic ulcer, and vomiting was the predominant symptom in some. Hematemesis and achlorhydria were of note in polyposis of the stomach (22 and 19).

There was practically unanimous agreement that anemia of the secondary type was the most common sign. Occult bleeding was declared greater in benign gastric tumor than with peptic ulcer, while gross hemorrhage was not rare. Some of these lesions were entirely symptomless. In the series of Balfour and Henderson, the youngest patient was 8, the oldest 69, and the average age was 46 (2).

The recognition that most benign tumors of the stomach are circumscribed filling defects, usually situated on the gastric walls, leaving the curvatures regular and free for the peristaltic movement, has been a basic aid to their diagnosis. The rugæ are obliterated in the area of the tumor, but immediately surrounding the tumor they are practically normal in pattern. Benign tumors do not reveal a niche, incisura, or any other evidence of spasm. Gastric retention is rare except when the lesion is at, or near, the pylorus.

When polyposis is extensive, there are small indentations along one or both curvatures. If the polyps are closely set or the barium too thick, the mixture will not penetrate to the gastric wall and the appearance will be broken and irregular. The differential diagnosis should include simple hypertrophy, food particles, gas in the transverse colon, crenulation of the greater curvature, gastric carcinoma, and hair ball (22 and 32).

Syphilis of the stomach results in lessened flexibility and mobility, contraction of varying degree, and absence of peristalsis. The pylorus tends to be gaping. There is no palpable mass and the patient is usually in good general condition compared to the extent of his gastric pathology (28).

Malignant tumors of the stomach, excluding carcinoma, are necessarily mesoblastic, fundamentally sarcomas. Ewing's (15) widely quoted estimate that these are 1 per cent of gastric tumors is generally accepted. Individual series vary moderately from this figure.

Sarcomas have occurred at all periods, from early childhood to advanced age (17). Balfour and McCann's (4) reported average age of 43 years for sarcoma as compared with 61 for carcinoma appears reasonably typical. Cheever's (9) series of lymphosarcoma having an age average of 53.5 suggests a later incidence for that type. In

Fig. 2.-*A*.

Fig. 2-*B*.

Fig. 2. Case 1. (*A*) Infiltrative lymphosarcoma, most pronounced in the proximal half of the stomach. The fundus, not directly involved, is compressed by the adjacent flexible tumor. (*B*) Supine view, showing greater distention of the uninvolved fundus and of the remaining involved proximal part of the stomach. On other films, a preserved interval between splenic flexure and stomach outlined the diffusely thickened walls of the stomach.

lesions occurring before the fourth decade, the likelihood of sarcoma is significantly increased.

A sex predilection appears established by Askey's (1) 70 per cent males in 335 reviewed cases.

The symptoms usually resemble those of carcinoma. Cheever (9), listing typical symptoms of lymphosarcoma, says, "It cannot be denied that this description fits perfectly a typical case of gastric carcinoma." There may be such localizing signs as epigastric pain, local tumor, vomiting, gastric bleeding, anacidity, and dyspepsia. The symptoms may be such nonlocalizing ones as weakness, loss of weight, fullness after meals, belching, and sour stomach (4); also they may be very vague (16, Case 1). Some have stressed a well-preserved nutrition (11).

While absent or inconspicuous pain is stressed by some (16 and 17), more outstanding pain is frequently reported (5, 6, and 36). In keeping with the ulcerations frequent in sarcoma, it may be ulcer-like (36 and 4) and of short duration, compared with that of benign ulcer (17). Severe pain, not usual in carcinoma (5), may be suspicious.

The duration of symptoms is quite variable. Balfour's series (4) gives an average of 18 months with a maximum of 9 years. Others record long continued symptoms or repeated similar attacks (30, 16, and 7). Malignant degeneration of tumors, originally benign, or exceptional low malignancy and slow growth, may be a factor.

Gastric sarcomas vary widely in histologic type, gross morphology, and roentgen findings. They have been classified morphologically as those growing intramurally, endogastrically and exogastrically, a grouping which has definite bearing on roentgen manifestations.

Ewing (15) classifies them pathologically as:

(1) Spindle-cell myosarcomas
(2) Lymphomatous tumors
  (*a*) as a part of the leukemic process;
  (*b*) in gastro-intestinal pseudoleukemia;
  (*c*) as part of a general lymphosarcoma;
  (*d*) in the form of localized lymphosarcoma.
(3) Miscellaneous sarcomas, variously described as alveolar, mixed cell, or angiosarcoma, or myxosarcoma.

One would infer that in Ewing's group of spindle-cell myosarcoma there would appear tumors named elsewhere as spindle-cell sarcoma, fibrosarcoma, some of the "round-cell" sarcomas, and some of the angiosarcomas.

Two tables of incidence of the varieties of gastric sarcoma will illustrate both their relative frequency and the terminology under which they are found.

Balfour's series of 45 cases (4)

Lymphosarcoma	32
Fibrosarcoma	5
Myosarcoma	3
Angiosarcoma	1
Perithelial angiosarcoma	1
Spindle-cell sarcoma	1
Sarcoma	6

D'Aunoy's and Zoeller's survey (10)

Lymphosarcoma	39
Round-cell sarcomas	24
Spindle-cell sarcomas	7
Fibrosarcomas	7
Round- and spindle-cell sarcomas	2
Leiomyosarcomas	2
Fibromyxosarcoma	1
Carcinosarcoma	1
Undifferentiated sarcoma	1
Sarcoma originating in reticulo-endothelial cells	1
Fibroneurosarcoma	1
Fibromyosarcoma	1
Mixed-cell sarcoma	1
Angiosarcoma	1
Fibrochondrosarcoma	1
Mixosarcoma	1
Polymorphous-cell sarcoma	1

LYMPHOBLASTOMAS

Lymphoblastomas, excluding the benign irritative hyperplasias, may be classified after Ewing (15) as follows:

(1) Lymphomatous group, including lymphatic leukemias, pseudoleukemias, and malignant lymphocytoma.
(2) Reticulocytomas, including myeloid leukemia, Hodgkin's disease, and large round-celled sarcoma.
(3) Primary endothelioma.

It appears best, in view of the pathologic confusion in this field, to consider them as lymphoblastoma. If one may add to primary lymphosarcoma the other clinically malignant lymphoblastomas, then this type of lesion, relatively radiosensitive, will include more than half of the non-carcinomatous malignant tumors of the stomach. This appears permissible for the present purpose although it may include lesions not technically sarcomas.

These arise, usually multicentrically, in submucosal lymphatic tissue. They involve the submucosa first, spreading to mucosa muscularis, and, sometimes, later, the serosa. The stomach may be extensively or locally involved. The gastric lesion may be part of a generalized involvement, in which case superficial glands are often available for biopsy, or the disease may be primary in the stomach. These lesions are primarily intramural though there may be an associated endogastric or exogastric mass.

Varied types of deformity are seen roentgenographically. A diffusely infiltrating form is characterized by a moderate decrease in caliber, thickened walls, diminished flexibility and peristalsis, and enlarged or distorted rugæ (33). This may be extensive or local (Fig. 2).

Such lesions are readily missed especially if represented only by a local stiffening of walls (33), or when limited to the cardiac half of the stomach. Here, a moderate constriction with prominent rugæ may be attributed to pressure from adjacent organs. A soft mass is not readily palpated, and sluggish or absent peristalsis is not remarkable. Thickened walls may register only as a preserved interval between the stomach and adjacent colon, or a pressure deformity of the latter (Case 1).

Such diffuse lesions are particularly in keeping with primary lymphosarcoma.

Multiple lobulated endogastric masses provide another type of lymphoblastoma. These masses are outlined by a reticulum of barium filling the interlobular crevices (33). Such manifestations suggest the less invasive forms, such as leukemia (25).

Another form is a rounded or irregular local mass encroaching upon the gastric lumen (5, 6, 11, 17, 29, and 33). Penetrative niches may occur in a large intragastric mass (6, 8, and 11). Rugal enlargements and distortions may overlie the diffuse lesions or be adjacent to ulcerative or massive lesions (5, 29, and 33).

Usually, constrictive lymphoblastoma has been said to occur only in the prepyloric regions (33); elsewhere, the delicate connective tissue framework of these tumors appears not to encourage this deformity (9). At least one exceptional midgastric encircling lymphosarcoma has been reported (21). Irregularity of the prepyloric constriction is stressed. Flexibility of the lesion is partially preserved. Peristalsis may or may not be seen and mass may or may not be palpable at the deformity. Shallow ulceration or even perforation may occur but a demonstrable niche is not featured (5, 33, and 18).

ROUND-CELL SARCOMAS

These are confused groups—D'Aunoy and Zoeller found them the second largest class reported. Balfour discarded three such cases as being carcinoma, leaving none so listed in his series. Ewing concludes that small round-cell sarcomas are, with rare exception, either lymphosarcoma or small round-cell carcinoma. The large round-cell sarcomas he divides among endotheliomas, tumors of lymphoid cells, carcinomas, and sarcomas of which the cells are not strictly round. This, if essentially correct, would accentuate still further the predominant position of lymphoblastoma. It would be natural that round-cell sarcomas tend to infiltrate intramurally (30) and resemble lymphosarcoma.

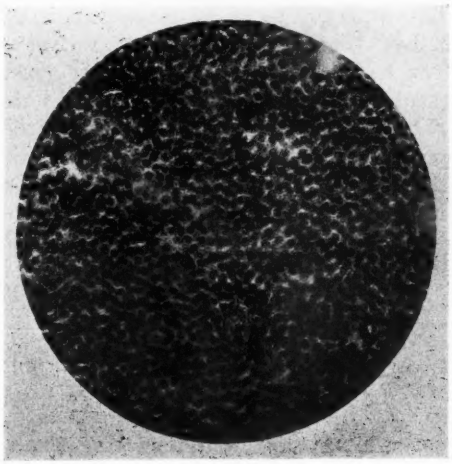


Fig. 3. Case 1. Lymphoid cell lymphosarcoma.

MISCELLANEOUS SARCOMAS

Concerning these, Ewing says, "None of these histologic features deserve special recognition as a separate variety of gastric tumor." No gross morphologic or roentgen characterization of this group can be attempted here.

OTHER NON-LYMPHOBLASTIC SARCOMAS

Most of these tend to grow either endogastrically or exogastrically instead of intramurally. In either case they may be pedunculated and mobile. Histologically, they may be fibro-, neurofibro-, myo-, or angiosarcoma, and appear to correspond to Ewing's spindle-cell myosarcoma group. They are radioresistant as a rule, and are more likely to be operable than their size would indicate (4 and 9), due to their non-invasive, sharply circumscribed character.

Growing endogastrically, they produce spheroidal or rounded defects, sharply demarcated from the adjacent uninvolved stomach (9, 36, and 7). Necrosis and hemorrhage are common, frequently with roentgenographic demonstration of a barium-filled button (Fig. 7) or deep penetrative niche (17 and 36) at the surface of the lesion.

Growing exogastrically, palpable masses, usually large, displace or distort the stomach (36 and 30). These may grow from the anterior or posterior walls and are particularly prone to extend between the layers of the gastro-hepatic or gastro-colic omentum (9). Such lesions are prone to hemorrhagic or cystic degeneration. Leiomyosarcoma, particularly, may be of the dumb-bell type, growing both intra- and exogastrically (36 and 23).

TYPES OF GASTRIC DEFORMITIES

One may list the types of gastric deformities which have been produced by non-carcinomatous tumors, considering other lesions which they may simulate.

Isolated rounded endogastric masses
- Adenoma or polyp
- Carcinoma *in situ* (5)
- Benign mesoblastic tumors, as,
  - fibroma
  - neurofibroma
  - leiomyoma
- Non-lymphoblastic sarcoma, as,
  - fibrosarcoma
  - neurofibrosarcoma
  - leiomyosarcoma (Fig. 6)
  - angiosarcoma (Fig. 7)
- Lymphoblastoma

The small rounded endogastric tumors, without alteration of peristalsis or of the adjacent gastric wall, can be accepted as positively benign only by operation and microscopic proof. Their appearance is not distinctive between the various types, all being potentially malignant. Hemorrhage, with this type of lesion, is not distinctive but the barium-filled niche speaks for non-lymphoblastic sarcoma, especially if associated with repeated hemorrage (7). These features were used successfully by one of us (D. R. L.) to diagnose a leiomyosarcoma (Fig. 7). The surface of the tumor may be "scalloped" in either the benign or malignant forms.

Excepting lymphoblastoma and carcinoma, tumors should not deform the adjacent part of the stomach or inhibit its peristalsis. An adjacent or general rugal hypertrophy speaks for the radiosensitive lymphoblastoma (29). The other types are radioresistant and not appropriate for the test of irradiation.

Polypose manifestations, local or general
- Polyposis
- Carcinoma
- Lymphoblastoma

The individual nodes of a benign polyposis should usually be smaller and more regular than those of lymphoblastoma or carcinoma; carcinoma should be more readily palpable, more rigid than either, and inhibits peristalsis. The response to moderate irradiation should identify lymphoblastoma (33).

Rugose manifestations, local or general
- Benign hypertrophy
- Rugal hypertrophy or ruga-like scars about benign ulcer
- Infiltrative carcinoma
- Lymphoblastoma (Fig. 4)

Benign rugal hypertrophy, local or general, may closely resemble lymphosarcoma. Both reduce peristalsis and flexibility (33 and 35). Lymphoblastoma is more apt to produce a moderate general decrease in caliber but may enlarge it grossly (29). The concentric scarring or irregular rugal deformities associated with benign ulcer and the meniscus deformity about carcinomatous ulcer are usually reasonably characteristic but may be simulated by lymphoblastoma (5). Occasional rugal types of carcinoma may resemble lymphoblastoma or benign hypertrophy (12). The test of irradiation may be useful.

Diffuse decrease in caliber, local or general
- Scirrhous carcinoma
- Syphilis
- Lymphoblastoma

Rigidity, a palpable mass, marked decrease in caliber, and absence of peristalsis would indicate carcinoma. Partial preservation of flexibility and peristalsis with rugose manifestations and moderate decrease in size suggests diffuse lymphoblastoma. No palpable mass, some flexibility, and absent peristalsis favors the diffuse type of syphilis (28).

While the decrease in size of the stomach is usually moderate (33 and 18), it may be extreme (18).

Irregular endogastric mass
- Carcinoma
- Lymphoblastoma
- Round-cell sarcoma
- Other non-lymphoblastic sarcomas

Any preserved flexibility or peristalsis may favor lymphoblastoma. The general bulkiness of sarcoma (11, Grier in discussion) may help. Again it is important to remember that large sarcomas may be removable (4 and 9). An apparent gross irregular intra-gastric growth may resolve roentgenographically under adequate pressure into voluminous rugal enlargement (Fig. 4). This speaks for lymphoblastoma.

Prepyloric constriction
- Carcinoma
- Benign ulcer, particularly the saddle variety with spasm
- Syphilis
- Inflammatory constrictions and retractions of perigastric origin
- Spasm, reflex
- Lymphoblastoma

The rigid, irregular, palpable lesion with absent peristalsis, anacidity and obliterated rugæ should be the usual carcinoma. The contracted prepylorus of niche-less ulcer should be not entirely inflexible, have visible rugæ, and be associated with gastric acidity. The reflex spasm may be decreased or removed by belladonna. History of upper abdominal infection or surgery may help with the perigastric inflammatory constriction. Absence of palpable mass, smooth annular constriction, some flexibility, absent local peristalsis, minor symptoms, and a positive serology speak for syphilis and justify the therapeutic test.

All that can be sought is some reason to suspect the occasional prepyloric lymphoblastoma. A marked irregularity, partly preserved flexibility, and peristalsis, and evident adjacent rugal hypertrophy may suggest that disease. Severe pain would suggest either ulcer or lymphoblastoma. A definite niche appears less likely than shallow ulceration in lymphoblastoma of constrictive type (5 and 33).

PRESSURE DISPLACEMENTS AND DISTORTIONS OF THE STOMACH

Non-gastric masses include the following:

- Tumors or enlargement of the spleen
- Tumors of the kidney, pancreas, adrenals, liver or retroperitoneal structures
- Massive metastasis from a concealed primary tumor
- Inflammatory masses

Exogastric sarcoma

Massive metastasis from an inconspicuous gastric lymphosarcoma (9)

The first group, too varied for complete listing, is an every-day occurrence. The occasional exogastric sarcoma should be remembered. Its connection with the stomach may be disguised by a pedicle which makes it mobile. On the other hand the sarcoma may be the dumb-bell type or cause such sharply localized pressure that a roentgen conclusion of endogastric or exogastric growth is difficult. Most exogastric sarcomas will be of non-radiosensitive varieties but may be operable though large. The stomach should be searched for signs of a gastric infiltration from which to suspect exogastric mass or metastatic lymph glands of the radiosensitive gastric lymphoblastoma.

COMMENT

A sharply circumscribed rounded endogastric mass bearing a demonstrable ulcerative niche may be considered probably a radioresistant non-lymphatic sarcoma. Such lesions without niche and not too large may be considered presumably, but only presumably, benign. Otherwise no roentgen signs even presumptive in value have been encountered by which to recognize non-carcinomatous tumors of the stomach. Only grounds for suspicion are found otherwise.

The radiosensitive lymphoblastoma should be suspected in lesions, rugose polypose, diffusely or irregularly infiltrative, and in cases in which preservation of

flexibility or peristalsis are otherwise not to be expected. The test of irradiation is to be considered in such cases. Study of the blood, roentgen examination of the chest, and thorough examination of superficial lymphatic glands is indicated because of the possibility of systemic lymphatic disease.

Methods of irradiation have not been discussed here. Holmes has warned that disastrous necrosis may be produced in lymphoblastoma (8). Pack and McNeer (29) have used intensive fractional irradiation with success in lymphosarcoma (29).

Upper abdominal mass displacing the stomach will occasionally be due to gastric sarcoma. The radiosensitive lymphoblastoma is suggested by associated infiltrative deformities in the stomach. The radioresistant sarcomas are possible when intrinsic gastric deformity is absent or is of the circumscribed rounded type.

An exceptional bulky tumor of the stomach suggests sarcoma.

Gastroscopy, when its possibilities are fully exploited, may be a great aid in the identification of gastric tumors.

CASE REPORTS

Case 1. A white married female (L. C.), aged 46 years, entered the hospital on Nov. 22, 1930, complaining of loss of ten pounds in three months, weakness, a questionable swelling in the left upper quadrant for one month, and more recently a dragging sensation in the abdomen while in the standing position. Appetite had been good, and stool habits normal. Some examiners denied, and others felt, a mass in the left upper quadrant. It was described as pulsating, freely movable, and having a rubbery edge. The red blood cell count was 4,300,000, hemoglobin 50 per cent, and white count 12,000 with normal differential. Roentgenographically, the stomach was low, vertical, and hypotonic, with no filling defect noted. There was some stasis in the duodenal cap. Peristalsis and mobility appeared not remarkable. The upper half of the stomach appeared constricted in the prone position but expanded to practically normal in the supine. (Fig. 2.) A barium enema demonstrated a defect near the splenic flexure, suggesting external pressure. Surgery disclosed a sarcoma of the stomach involving all the stomach from two inches below the cardia, and also the anterior pole of the great omentum. The stomach was resected and the patient died the following day.

The specimen of stomach examined pathologically was 21 cm. in length and 11 cm. in diameter at the fundus. The outer surface of the stomach, especially the proximal two-thirds, was covered with a gray tumor growth. The wall was greatly thickened, being 3 cm. in the fundus. The microscopic diagnosis was lymphosarcoma of the stomach. Dr. Newton Evans, in review, considers this to be of lymphoid cell type. (Fig. 3.)

Comment.—Infiltrative type of lymphosarcoma. Contributing to the failure to recognize it roentgenologically were the following: marked flexibility of the gastric walls even at the site of greatest infiltration; preservation of peristalsis throughout; too ready acceptance of external pressure as a cause for the manifestations observed. It is noteworthy that the growth was described as "covering" the stomach. We hope that so classical an infiltrative lymphoblastic lesion would not be missed now.

Case 2. A white married female (M. B.) aged 63 years, had dull constant pain, unrelated to meals, in the left upper quadrant for ten months. In the past ten weeks there had been a swelling of this region and the pain had become more severe. Weakness, anorexia, and loss of 15 pounds were noted in the four months before entrance. On physical examination there was noted a large, firm, sharp-edged mass in the left upper quadrant running obliquely across the umbilicus. The mass moved with respiration and was tender to pressure. The liver was not palpable. There was no free acid, and the combined acidity was 12 by the Ewald test-meal. There was 4 plus occult blood in the stools.

The red blood cell count was 3,780,000, the white count 18,000 with 90 per cent polymorphonuclears. The roentgenologic

Fig. 4-*A*. Case 2. Lymphosarcoma. Irregular encroachment on the lumen of the stomach, coarsely rugose, but because of its flexibility and hard adjacent extragastric mass, interpreted as pressure distortion.

examination demonstrated poor filling of the upper half of the stomach; the barium passed rapidly into the duodenum. (Fig. 4.) On palpation, there was noted a hard, well-defined mass lying medially and extending posteriorly to the body of the stomach. The incomplete filling appeared to be due to pressure defect of the tumor rather than to a primary gastric tumor. At surgery a large mass occupied the upper left half of the abdomen. There were extensive dense adhesions. The stomach was smooth and the walls greatly thickened. The patient died the following day.

Autopsy disclosed a white glistening gastric neoplasm which infiltrated the walls and involved the entire stomach except the cardiac portion and the pyloric ring. There were no ulcerations of the mucosa. There was extension along the dome of the diaphragm, the left lobe of the liver, and the gastro-splenic ligament. Microscopically the tumor was a lymphosarcoma of the stomach. Dr. N. Evans reviewed this material and concluded that the lesion was of the reticulum cell type. (Fig. 5.)

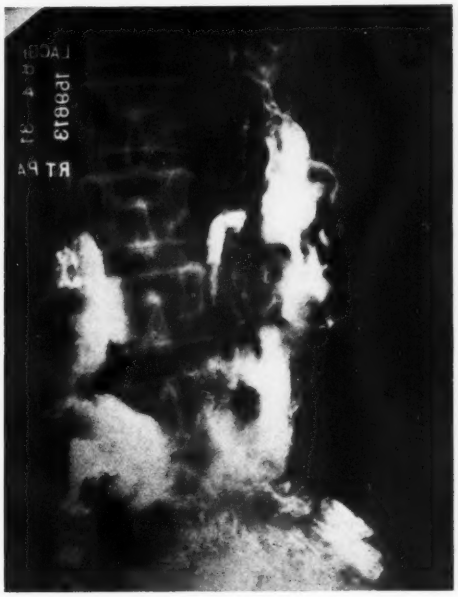


Fig. 4-*B*. Case 2. Better demonstration of the rugose manifestations and flexibility of the stomach.

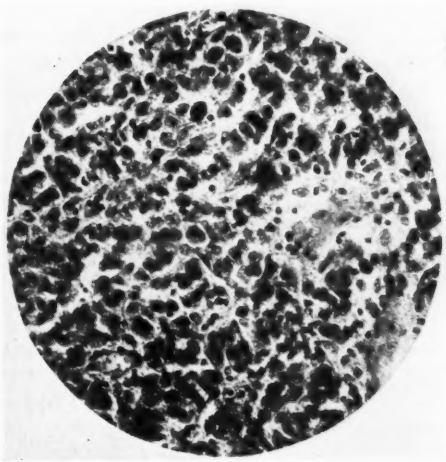


Fig. 5. Case 2. Reticulum cell lymphosarcoma.

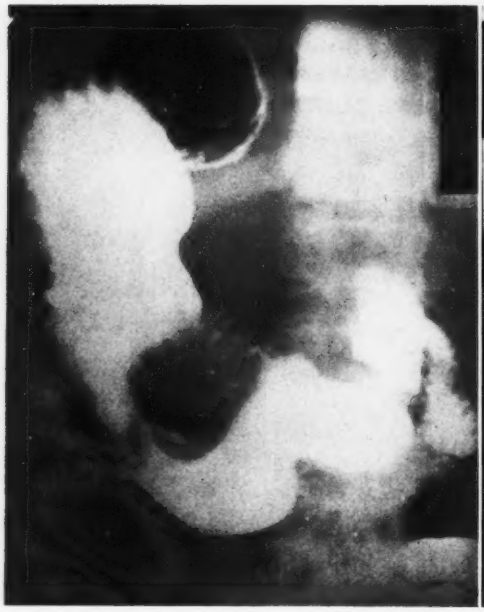


Fig. 6. Case 3. Angiosarcoma, endogastric and exogastric.

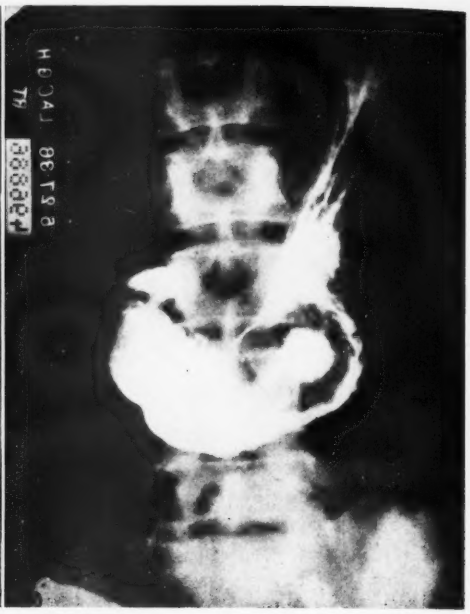


Fig. 7. Case 4. Leiomyosarcoma, endogastric, peristalsis throughout; crater of ulcer demonstrated.

Comment.—Infiltrative lymphosarcoma with marked extragastric extension. Less ready acceptance of rugose manifestations and distortion of the stomach as due to pressure would now give grounds for suspecting lymphoblastoma. It is noteworthy that the exogastric mass was hard to palpation while the stomach was quite flexible.

Case 3. A white male (F. B.), aged 51 years, entered the hospital first in June, 1931, in a restless, non-co-operative state bordering on a dementia. The patient apparently had been well until two weeks before entrance when he began to have pains in his abdomen, loss of appetite, sensation of fullness, nausea, and occasional attacks of vomiting. There was also some loss of weight and strength. Physical examination was essentially negative. The Ewald test-meal disclosed free HCl of 4 and a combined acidity of 7. The entering blood count was 1,900,000 red blood cells, hemoglobin 45 per cent, white cells 15,400, with 82 per cent polymorphonuclears. A series of blood transfusions finally brought the red count up to 3,500,000 and the hemoglobin to 60 per cent. The stools were tarry. Roentgenographically, the stomach revealed a large rounded defect in its mid-portion which gave the impression of extrinsic pressure from the anterior side. (Fig. 6.) Peristalsis passed throughout the stomach. There was no retention. The colon was negative. Surgically, there was an apparently benign tumor of the anterior and superior surface of the stomach, predominantly angiomatous. The mass was about 3 × 4 × 2 inches. Two ulcerated areas showed the site of hemorrhages into the stomach. Vascularity about the stomach was greatly increased. Attached to this mass was a pedunculated nodule about 4 cm. in diameter composed of soft gray tissue and somewhat hemorrhagic. The mass was excised. Pathologically, two ulcerated areas, 2 cm. in diameter, were observed. Subadjacent to the mucosa was a large soft mass about 8 cm. in its greatest diameter composed of very soft gray tissue resembling sarcoma. Microscopically, the lesion was an angiosarcoma of the stomach. In a recent review of the microscopic section, Dr. Newton Evans

was of the opinion that the lesion was an angioendothelioma which would be essentially the same as angiosarcoma. However, the possibility of an atypical carcinoma was to be considered. The patient re-entered the hospital a year and a half later complaining of swelling of the abdomen and obstipation for two weeks. Physical examination revealed a prominent abdomen with bulging in the flanks and a large indefinite mass in the mid-line. The red blood cell count was 4,400,000 and hemoglobin 60 per cent. An abdominal paracentesis was done and 3,000 c.c. of bloody fluid was obtained. The patient was discharged to the County Infirmary as an inoperable case.

Comment.—While the description of gross pathology is not entirely clear, the lesion appears to be of dumb-bell type, the extrinsic mass serving to disguise the endogastric protrusion. The local persistence of peristalsis indicates a non-infiltrative lesion such as may be expected from non-lymphoblastic sarcoma, including myo-, fibro-, and at least some angiosarcomas. The niche, if demonstrated, would have spoken against a benign lesion and for sarcoma.

REFERENCES

(1) ASKEY, E. V., HALL, E. M., and DAVIS, K. S.: Sarcoma of the Stomach. West. Jour. Surg., 1930, **39,** 830.

(2) BALFOUR, D. C.: Lesions of the Stomach and Duodenum. Surg. Clin. No. America, 1926, **6,** 1175–1183.

(3) BALFOUR, D. C., and HENDERSON, E. J.: Benign Tumors of the Stomach. Am. Surg., 1927, **85,** 354–359.

(4) BALFOUR, D. C., and MCCANN, J. C.: Sarcoma of the Stomach. Surg., Gynec. and Obst., 1930, **50,** 948–953.

(5) CABOT, R. C.: Case Records of the Massachusetts General Hospital, Cases 19,401 and 19,402. New Eng. Jour. Med., Oct. 5, 1933, **209,** 700–705.

(6) Idem: Case Records of the Massachusetts General Hospital, Case 19,222. New Eng. Jour. Med., June 1, 1933, **208,** 1167–1169.

(7) Idem: Case Records of the Massachusetts General Hospital, Cases 21,111 and 21,112. New Eng. Jour. Med., 1935, **212,** 481–488.

(8) Idem: Case Records of the Massachusetts General Hospital, New Eng. Jour. Med., Nov. 22, 1934, **211,** 976–979.

(9) CHEEVER, D.: Clinical Aspects and Treatment of Primary Lymphosarcoma of the Stomach and Small Intestines. Ann. Surg., 1932, **96,** 911–923.

(10) D'AUNOY, R., and ZOELLER, A.: Sarcoma of the Stomach. Am. Jour. Surg., 1930, **9,** 444–464.

(11) DRANE, R.: A Brief Consideration of Sarcoma of the Stomach. Am. Jour. Roentgenol. and Rad. Ther., 1935, **34,** 755–758.

(12) EISLER, F.: Erroneous Findings in X-ray Diagnosis of Stomach. Year Book of Radiology, 1936, pp. 249–251. Year Book Publishers, Inc., Chicago.

(13) EUSTERMAN, G. B., and BALFOUR, D. C.: The Stomach and Duodenum. Saunders, Philadelphia, 1936.

(14) EUSTERMAN, G. B., and SENTY, E. G.: Benign Tumors of the Stomach. Surg., Gynec. and Obst., 1922, **34,** 5–15.

(15) EWING, J.: Neoplastic Diseases. Saunders, Philadelphia, 1928.

(16) FREEMAN, D. B.: Fibroblastic Sarcoma of the Stomach. Am. Jour. Surg., 1934, **25,** 543–545.

(17) HAGGARD, W. D.: Sarcoma of the Stomach, with Report of a Case and an Analysis of 107 Cases Operated upon. Surg., Gynec. and Obst., 1920, **31,** 505–511.

(18) HOLMES, G. W., DRESSER, R., and CAMP, J. D.: Lymphoblastoma: Its Gastric Manifestations, with Especial Reference to Roentgen Findings. RADIOLOGY, 1926, **7,** 44–50.

(19) JOYCE, T. M., and DIACH, S.: Gastric Polyposis. Surg. Clin. No. America, 1933, **13,** 1417–1422.

(20) JUDD, E. S., and HOERNER, M. T.: Benign Tumors of the Stomach. Am. Jour. Surg., March, 1936, **31** (new series), 427–431.

(21) KEIJSER, S.: Sarcoma of the Stomach and its X-ray Diagnosis. Year Book of Radiology, 1936, pp. 255–257. Year Book Publishers, Inc., Chicago.

(22) KIRKLIN, B. R., and BRODERS, A. C.: Gastric Polyposis. Jour. Am. Med. Assn., 1932, **98,** 95–99.

(23) LAMSON, O. F.: Sarcoma of the Stomach. Surg. Clin. No. America, 1933, **13,** 1429–1432.

(24) LOCKWOOD, B. C.: Benign Tumors of the Stomach. Jour. Am. Med. Assn., 1932, **98,** 969–973.

(25) MEAD, C. H.: Chronic Lymphatic Leukemia Involving the Gastro-intestinal Tract. RADIOLOGY, 1933, **21,** 351–365.

(26) MEYER, K. A., and ROSI, P. A.: Leiomyoma of the Stomach. Surg. Clin. No. America, 1933, **13,** 1245–1249.

(27) MOORE, A. B.: Roentgenologic Study of Benign Tumors of the Stomach. Am. Jour. Roentgenol. and Rad. Ther., 1924, **11,** 61–66.

(28) MOORE, A. B., and AURELIUS, J. R.: Roentgenologic Manifestations in Eighty-seven Cases of Gastric Syphilis. Am. Jour. Roentgenol. and Rad. Ther., 1928, **19,** 425–432.

(29) PACK, G. T., and MCNEER, G.: Sarcoma of the Stomach: A Report of Nine Cases. Ann. Surg., 1935, **101,** 1206–1224.

(30) RANSOHOFF, J. L., and DICKSON, T. R.: Sarcoma of the Stomach. Ann. Surg., 1933, **97,** 68–73.

(31) REINIETS, J.: The Frequency and Pathologic Aspects of Gastric Leiomyoma (unpublished thesis). Proc. Staff Meetings, Mayo Clinic, 1930, **5,** 363.

(32) RIGLER, L. G., and ERICKSEN, L. G.: Benign Tumors of the Stomach. RADIOLOGY, January, 1936, **26,** 6–15.

(33) RUGGLES, H. E., and STONE, R. S.: Lymphoblastoma Involving the Stomach Roentgenographically Considered. Calif. and West. Med., 1930, **33,** 486–490.

(34) SUTHERLAND, C. G.: Polypoid Tumors in the Pyloric End of the Stomach. RADIOLOGY, 1926, **6,** 520–523.

(35) WINDHOLZ, F.: Roentgenologic Diagnosis of Hyperplasias of the Gastric Mucosa. RADIOLOGY, 1931, **17,** 514–519.

(36) YARDUMIAN, K.: Primary Gastric Sarcoma. Arch. Path., 1935, **20,** 590–595.

DISCUSSION

DR. ROBERT S. STONE (San Francisco): Dr. Carter's paper is of such a complete nature that it is one we will all want to have for reference. I wish to commend Dr. Carter on the method of attack which he has taken on this problem, a method which is all too infrequently taken by us as roentgenologists. Due to our previous medical training and the influence of the medical colleagues who surround us, we nearly all attack problems from the pathologist's or clinician's standpoint. Dr. Carter has taken the true method of the roentgenologist, namely, he has taken up the various deformities and discussed what lesions might cause each type of deformity.

After all, that is our daily problem. We are not confronted with the problem of diagnosing the pathologic condition from the microscopic slide or the bedside findings. We must diagnose the pathologic condition from the deformities that we see on the films or fluoroscopic screen. I wish that more of us would take Dr. Carter's line of attack on the problem when presenting a paper dealing with diagnosis.

He has covered the problem so thoroughly that there is little left to be said in the way of discussing the various lesions. I would like, however, to say in one or two words something about the prepyloric spasms which he mentioned briefly in his paper.

It brings up the problem which was brought up previously by Dr. Kirklin, namely, re-examination of these patients and the cause of the spasms.

We have had a number of cases in which, on re-examination without medication the spasm has disappeared. I am, rightly or wrongly, considerably impressed by the fact that in the re-examination with belladonna, atropine, benzadrine sulphate or any of the antispasmodics, it is not a question of the antispasmodic at all but of simply getting the patients back into the fluoroscopic room after they have been there once and know what it is all about. Maybe it is a question also of their being treated more gently the second time than they were the first time, and therefore the patient does not have the same reaction.

On the other hand, there are cases in which operations revealed no evidence of disease and yet in which the spasm was not relieved either by repeated examinations, even at some months' interval, or by the administration of any of the antispasmodics. I think that neither repeated examinations nor the administration of antispasmodics necessarily establishes whether some of these defects in the prepyloric region are caused by an actual organic lesion or by a localized spasm.

This holds particularly, I believe, for cases of gall-bladder disease. We are inclined to think at times that the deformities seen in such cases are due to adhesions and when the surgeon opens the abdomen and says he finds some adhesions between the gall bladder and the duodenum, we say, "Well, that must have been the cause of the deformity." But if you look back on your films you find your deformity was nowhere near where the adhesions were. The deformity was in the prepyloric region of the stomach and there was no pull of the adhesions on that point.

One other point I would like to emphasize is the differential diagnosis between polypoid tumors. Of course, if the stomach wall is rigid around a polypoid tumor, we have no hesitation in considering it as a malignant lesion with invasion of the stomach wall; but if the stomach wall is not rigid and peristalsis still passes through, we are inclined to diagnose it as benign polyposis.

I wish to emphasize what has been pointed out before, namely, that with polypoid lymphosarcoma in the early stages there may not be any invasion of the muscular wall and there may be absolutely no rigidity. So we come to the point that Dr. Carter made—that we must not consider these tumors as benign until they have been proven to be so. We have to accept them as malignant.

There is one other point which Dr. Carter made that I think is of great significance. That is, if you are able to demon-

strate an ulcer niche in the polypoid tumor, it is much more likely to be a malignant tumor than if you do not find any such niche on the polypoid growth. Dr. Templeton in his scientific exhibit demonstrates one case in which the appearance of niche was caused by a dimple in a benign polyp.

Let me again congratulate Dr. Carter on his thorough and lucid presentation of this subject from the roentgenologist's point of view.

DR. LEO G. RIGLER (Minneapolis, Minn.): I would just like to say a word or two about the incidence of benign tumors of the stomach. I think the 11 per cent figure which Dr. Carter mentioned is my responsibility, more or less, and I would like to emphasize it again.

We have looked over our autopsy figures very carefully, as has Stewart, and it is amazing what a high percentage of the tumors in the stomach appear to be actually benign. I think that in spite of the fact that they appear to be benign microscopically as well as from the typical roentgen characteristics which Dr. Carter so well described, we have to be very careful about the prognosis in those cases.

We have seen, ourselves, a number of them become malignant almost under our very eyes, by observation over a period of years, and, on the other hand, I have now

Fig. 1. Neurofibroma of stomach wall showing normal folds of mucous membrane over filling defect.

three cases, one of which I have followed for eight years, another one for four years, in which multiple tumors of this type have remained absolutely constant and have not changed their appearance whatsoever

I think the technic which Dr. Carter emphasized in the beginning of his paper is extremely important if we are to make the diagnosis of these small benign polypi, and I think the diagnosis is very important because certainly a large percentage of the

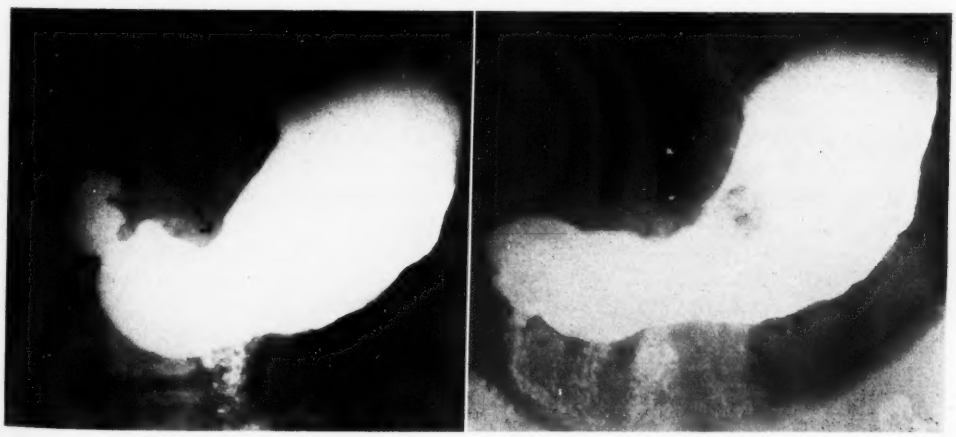


Fig. 2. Neurosarcoma of stomach. Roentgenogram made Jan. 16, 1933, simulates early carcinoma. Increase in size of lesion shown on second examination, Sept. 11, 1933.

malignancies of the stomach arise from such benign tumors and the diagnosis of benign polyp of the stomach may be the earliest possible diagnosis that we can make of a carcinoma.

I also want to bring out just one more point and that is with regard to the intramural tumors which Dr. Carter mentioned, particularly the neurofibroma. I would like to show just two slides, if I may bring that out.

The diagnosis of an intramural tumor may be beautifully demonstrated if we can show the defect and then the rugal pattern lying over the defect. This was a neurofibroma of the gastric wall, and we removed it at autopsy and it demonstrated beautifully why this appearance (Fig. 1) was present—the lesion extending outward to a large extent and simply displacing the mucosa over it without invading it and permitting the mucosal pattern to retain its normal appearance. Schatzki has called my attention to the fact that the demonstration of normal rugæ over a tumor defect does not always mean that the tumor is intramural because the mucosal pattern from the opposite wall may be producing the roentgen findings.

Some of these cases are malignant, as shown in Figure 2. We saw this patient at this time, knowing nothing else about the history. We found a small area of infiltration on the lesser curvature and I was very proud of the diagnosis of early carcinoma of the stomach. It was later discovered that the patient had some tumors in the lung, which certainly did not look like the ordinary metastasis, but in view of the diagnosis of carcinoma were considered to be metastasis—no surgery was attempted.

I saw the patient some seven months later and you see the change in the appearance with the invasion of a tumor extending into the stomach and which is beginning to take on the characteristics of a typical malignant lesion.

This proved to be a neurofibrosarcoma, such as Dr. Carter mentioned, and these lesions in the lungs were neurofibromas which had undergone malignant degeneration and had become neurofibrosarcoma. This is not as uncommon a tumor as you might think. Neurofibromatosis is not unusual and the tendency is to involve the stomach in a considerable number of cases.

HERNIA OF THE CARDIAC END OF THE STOMACH THROUGH THE DIAPHRAGM[1]

By MAURICE F. DWYER, M.D., F.A.C.P., *Seattle, Wash.*

DURING the past decade many articles have been written on diaphragmatic hernias involving the cardiac end of the stomach, and in reviewing them one is impressed with the variations in the incidence of such hernias and in the clinical importance attributed to the various forms.

The term "diaphragmatic hernia" suggests to the majority of physicians a tear in the diaphragm, or a protrusion of a portion of a viscus through an opening in the diaphragm as the result of trauma. It may more accurately be defined, however, as a protrusion of abdominal contents through an abnormal opening in the diaphragm which has resulted from imperfect development, anatomic weakness, or trauma. Recent statistics show that diaphragmatic hernia due to trauma is uncommon, comprising less than one-third of the reported cases. These hernias are classified as approximately one-third congenital, a little more than one-third acquired after birth, and less than one-third traumatic.

The majority of diaphragmatic hernias follow no known injury, and consequently the relaxed or enlarged openings in the diaphragm, capable of permitting the protrusion of a viscus at some time during life, must be assumed to be congenital in origin. Imperfections of development, such as harelip, cleft palate, spina bifida, hypospadias, and relaxation of the inguinal rings, are well recognized. It is, therefore, not at all strange that a structure as complicated as the diaphragm fails at times to develop normally. Thus if prenatal fusion is at all imperfect, the strains to which this imperfectly developed structure is subjected in early life—such as birth trauma, crying, violent coughing, spasms, etc., all of which increase intra-abdominal pressure—may result in hemorrhagic infiltration in the diaphragm. The consequent fibrosis and stretching following the hemorrhagic infiltration may, in turn, result in herniation through a weakened area. While this condition may not be truly congenital, still it does occur in very early childhood.

The true incidence of diaphragmatic hernia is hard to determine, as there is such discrepancy in the literature; however, it is certain that at present many more cases are diagnosed than formerly, and occasionally a previously unexplained abdominal complaint on re-examination is accounted for by a protrusion of a portion of a viscus through an opening in the diaphragm. As this paper is to deal solely with hernia of the cardiac end of the stomach, other types of diaphragmatic hernia and abnormalities of the diaphragm will not be discussed.

Harrington (1), in discussing the incidence of diaphragmatic hernia as found in the Mayo Clinic over a period of 32 years, brought out the interesting point that during the latter eight years five times as many cases were diagnosed at the Clinic as in the preceding 24 years. In 1931, Hedblom (2) reviewed the literature and tabulated 1,435 cases. Of these, 190 (13.2 per cent) were hernias through the esophageal hiatus. Ritvo (3) found 60 cases of hernia through the esophageal orifice in 9,000 routine examinations, an incidence of 0.7 per cent of patients complaining of general gastric symptoms.

In comparing reports published during the past ten years with those published previously, there can be no doubt but that the incidence of diaphragmatic hernia, especially that through the esophageal hiatus, is increasing rapidly. This increase is due to the fact that the clinician is becoming more familiar with the symptoms suggestive of a diaphragmatic hernia, and is thereby directing his attention to the diaphragm, especially in the absence of objective findings

[1] Presented before the Radiological Society of North America at the Twenty-second Annual Meeting, at Cincinnati, Nov. 30–Dec. 4, 1936.

to account for the symptoms; and also to the fact that roentgenologists now are constantly keeping such a lesion in mind and demonstrated such a lesion, the clinician and roentgenologist must not then rest on their oars and consider that, having found

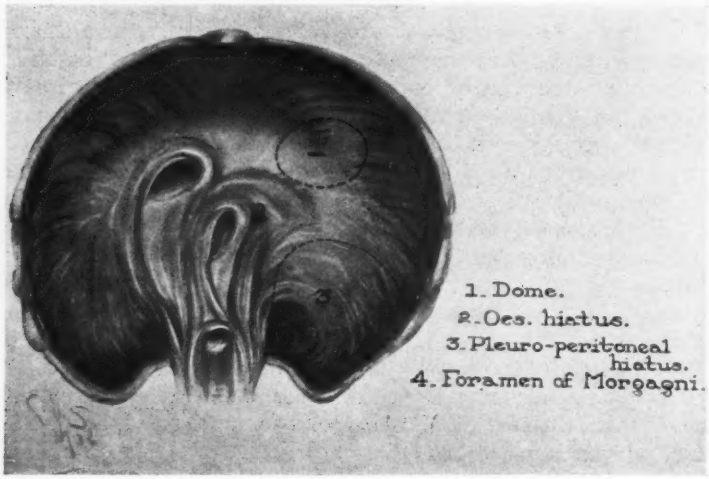


Fig. 1. Sites of diaphragmatic hernias.

are employing a technic of fluoroscopic examination necessary to demonstrate the hernia. If the roentgenologist does not himself elicit suggestive clinical signs and symptoms of the probable existence of a gastric hernia, the possibility of such a condition must be mentioned to him by the clinician, for esophageal hiatus hernias will nearly always, unless of large size, be overlooked if the patient is examined only in the vertical position.

We have a record of seven cases in a series of 6,500 routine gastro-intestinal examinations. It is obvious that in the past some cases have been overlooked because not sufficient clinical and roentgenologic importance was attributed to herniation of the cardiac end of the stomach as an entity causing gastric symptoms. On the other hand, the writer does not wish to convey the impression that these hernias are a relatively frequent source of gastric complaints, and that every protrusion of the cardiac end of the stomach through a diaphragmatic opening, no matter how small, is the cause of the symptoms for which the patient has sought relief. After having the hernia, they have made a complete diagnosis. To do so without further investigation may result in overlooking a diseased gall bladder, a gastric ulcer, or an esophageal lesion. However, a hernia of the cardiac end of the stomach, especially through the esophageal hiatus, may be the sole cause of the gastric symptoms and must be constantly borne in mind by both clinician and roentgenologist and recognized when present.

A simple and practical classification of diaphragmatic hernia is as follows: (1) congenital, either true or false; (2) acquired, either true or false, and (3) traumatic (Fig. 1). Harrington (4) states that the congenital and acquired types occur through (1) the dome of the diaphragm, (2) the esophageal hiatus, (3) the pleuroperitoneal hiatus, or foramen of Bochdalek, (4) the foramen of Morgagni, and (5) any embryonic fusion point of the diaphragm.

The traumatic type may be classified as (1) those due to direct injury, such as gunshot or stab wounds, which may occur at any point in the diaphragm, and (2) those due to indirect injury, which usually occur

at an embryologic fusion point. A hernia not due to trauma may, theoretically at least, occur at any of the normal openings eventually force some one or other of the abdominal organs into the thorax. The size of the opening varies considerably.

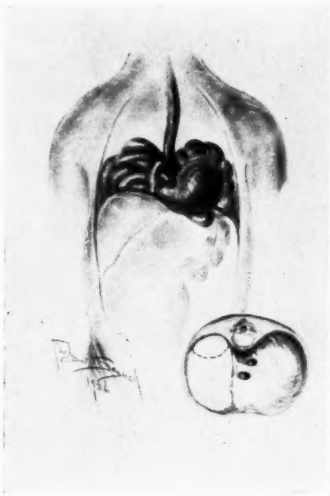


Fig. 2. Hernia through the pleuroperitoneal hiatus.

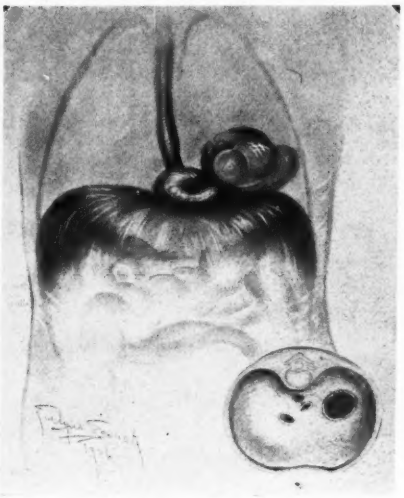


Fig. 3. Hernia through the dome of the diaphragm.

in the diaphragm, but actually most hernias of this group present themselves through an enlarged or relaxed esophageal opening. Herniations through the vena cava and aortic openings are unknown, and Healy (5) points out that this is true because the esophageal opening is the only muscular one in the diaphragm, the others being made up of tendinous attachments.

Hernia through the Pleuroperitoneal Hiatus, also called hernia through the costovertebral angle, is due to a failure of closure of the median and dorsal portion of the pleuroperitoneal membrane (Fig. 2). The passage remains open until the third month of intra-uterine life, and that on the left possibly closes later than that on the right.

The channel of communication between the pleural space and the peritoneal cavity is the pleuroperitoneal canal. If this canal should remain open through deficiency in the rate of growth and closure, then the pleural and peritoneal cavities will not be separated completely from one another and the positive intra-abdominal pressure will The pleuroperitoneal passage is small at the time the hernia occurs, being approximately the same size in relation to the rest of the abdominal wall as the orifice of a large inguinal hernia is in the adult. Complete absence of the left half of the diaphragm is an exaggerated form of this type of hernia.

Hernia through the Dome.—This hernia is an opening actually through the substance of the diaphragm and is independent of any of the natural openings (Fig. 3). It is due to rupture or destruction of a portion of the membranous diaphragm. The defect is more common in the left dome than in the right, which is accounted for by the protection given the diaphragm by the large right lobe of the liver. The position of the defect is fairly constant, though there may be considerable variation in its extent, and the aperture may be round or oval. A portion of the stomach and colon is invariably herniated, and occasionally the spleen and portions of the small intestine may be found in the thorax.

The presence of this abnormal opening is

difficult to explain. Hume (6) believes there are two possible explanations of the defect. The first is that there may be a delay or interference with the growth of the pleuroperitoneal folds, causing a failure to unite with the dorsal portion of the septum transversum, and that it is possible, therefore, that some temporary disturbance of nutrition may affect the relative rate of growth of the components of the diaphragmatic premordium. The second possibility is that the herniation may occur after the membranous diaphragm has formed at the time the pleural cavity is relatively unfilled, and a portion of the membranous diaphragm lies unprotected owing to the inequality of the size of the lobes of the liver at this stage of development.

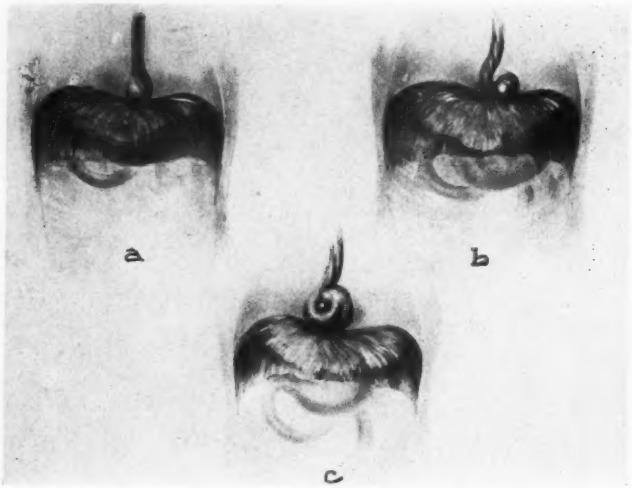


Fig. 4. Hernias through the esophageal hiatus. (*a*) Esophageal hiatus hernia with congenital shortening of the esophagus. (*b*) Esophageal hiatus hernia without congenital shortening of the esophagus, in which the esophagus does not form part of the hernia (para-esophageal hernia). (*c*) Esophageal hiatus hernia in which the esophagus is not shortened but in which the distal portion of the esophagus is a part of the hernial contents.

Herniation of a Portion of the Stomach through the Esophageal Hiatus is by far the most common type of diaphragmatic hernia (Fig. 4). Akerlund (7) has demonstrated that esophageal hiatus hernias are six or seven times as frequent as all other non-traumatic diaphragmatic hernias combined, and has classified these hernias on an anatomical basis into three groups.

1. Esophageal hiatus hernia with congenital shortening of the esophagus.
2. Esophageal hiatus hernias without congenital shortening of the esophagus, in which the esophagus does not form part of the hernia (*i.e.*, para-esophageal hernias).
3. Esophageal hiatus hernias in which the esophagus is not shortened but in which the distal portion of the esophagus is a part of the hernial contents.

Esophageal hiatus hernias of the last group are the most common, and those of the first group the least common in occurrence.

The size of the esophageal hiatus may vary considerably. Embryologically this defect is due to the fact that the development of that portion of the diaphragm derived from the dorsal mesentery and the mesoderm of the receding Wolffian body leaves not only a defect of varying extent in the muscular and tendinous structures but often leaves also a deformity in the conformation of the dome of the diaphragm and a shortening of the esophagus.

Esophageal hiatus hernias are essentially congenital in the same sense that hernias in the inguinal region are congenital. Due to a deficiency in embryological development there may be a congenital weakness of the esophageal ring, and, as in inguinal hernia, later on in life, through trauma, pregnancy, or any increase in intra-abdominal pressure, a definite hernia may develop. Many persons with abnormally relaxed esophageal hiati go through life without the slightest herniation of a viscus through the relaxed opening, and only after the increase of intra-abdominal pressure from some cause forces enough of the stomach or intestines through the opening to produce symptoms, is the condition suspected.

Harrington, in order to determine the variation of size of the esophageal ring, examined this opening in 500 cases during the course of other abdominal operations. The summary of his findings showed that in 65 per cent of cases the diaphragm was closed tightly around the esophagus, and he could determine no appreciable space between the two structures. In 35 per cent of cases one finger or more than one could be placed between the esophagus and the margin of the diaphragmatic opening. Of this group, in 55 per cent only one finger could be inserted between the esophagus and the diaphragm, in 40 per cent two fingers could be inserted, and in 5 per cent three fingers could be so inserted. In all the cases in which three fingers could be inserted, special roentgenologic examinations were made later and occasionally revealed a small hernia.

Hernia through the esophageal hiatus is the most frequent variety of diaphragmatic hernia found in adults and may exist for years without producing symptoms. These hernias as a rule present more uniform symptoms than hernias situated elsewhere in the diaphragm because usually the only organ involved is the stomach. The symptoms, therefore, are chiefly suggestive of a stomach lesion or a reflex condition producing gastric symptoms, and the severity of the symptoms generally depends upon the degree of herniation of the stomach.

There is no characteristic set of symptoms or syndrome diagnostic of this condition, and a pre-operative diagnosis can be made only by the roentgenologist. Hernias through the esophageal hiatus, however, do present symptoms some of which should make the clinician strongly suspicious of this condition. On the other hand, many symptoms, indefinite in themselves, merge into the general group of stomach complaints so commonly associated with various lesions of the upper abdomen. In reviewing the histories of such cases, the clinician must constantly bear in mind that a diaphragmatic hernia may be the cause of the complaint for which the patient seeks relief.

Harrington (8), emphasizing this, stated that the diaphragm should always be examined by the surgeon in all cases in which the conditions found at operation do not seem sufficient to account for the patient's symptoms, for experience has shown that some of the unsatisfactory results following operative procedures, especially on the stomach, gall bladder, and appendix, may be due to an unrecognized esophageal hernia. In recent years surgeons and roentgenologists have paid considerable attention to herniations of the stomach through the esophageal opening of the diaphragm, but on reviewing the literature on this subject we found that little has been contributed by internists, certainly nothing in proportion to what has been written concerning other conditions, the incidence and clinical importance of which are no greater.

The general symptomatology of the majority of patients presenting a herniation of a portion of the stomach through an esophageal opening in the diaphragm is usually mild at the beginning. Epigastric distress of varying degree occurs during or shortly after a meal. The severity of this distress is in proportion to the degree of herniation of the stomach and to the amount of food taken. Pain in the epigastrium is a frequent complaint, the pain varying in character from a localized dull aching distress in the lower mediastinum

and epigastrium to an excruciating pain referred to the back and mammary regions and radiating down the arms, accompanied by tachycardia and increasing when the patient assumes a horizontal position. The clinical picture of a severe attack may closely simulate that of coronary thrombosis.

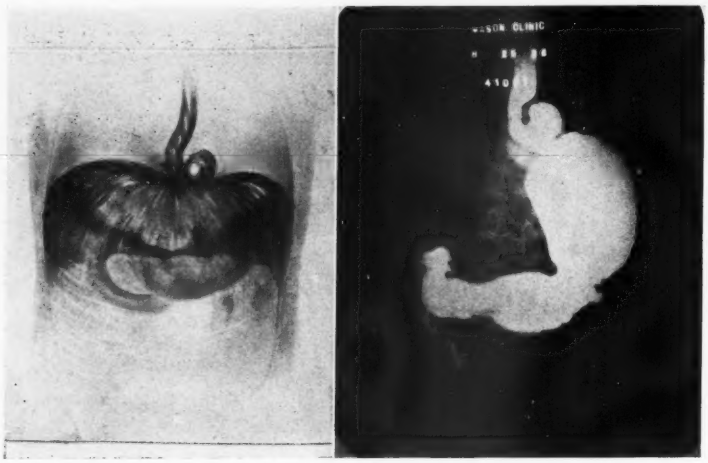


Fig. 5. Drawing and roentgenogram of Case 1, showing esophageal hiatus hernia of the para-esophageal type.

Pain due to the condition is often relieved by walking, bending, and the eructation of gas. Nausea and vomiting are commonly noted and occur from a few minutes to an hour after eating, depending on the degree of herniation.

Dysphagia is another relatively common complaint. The difficulty in swallowing may be temporary and mild, or it may be prolonged and so severe that an erroneous diagnosis of cardiospasm or other lesions of the lower end of the esophagus may be made.

Herniation through the esophageal hiatus may also be the cause of a previously unexplained secondary anemia. Bock (9) and his associates reported a series of 10 cases of secondary anemia due to intermittent bleeding from the portion of the stomach involved in the hernia. Clinical, operative, and postmortem examinations failed to demonstrate any lesion other than the hernia to account for the anemia. Their findings seem to warrant the conclusion that the congestion of the gastric mucous membrane due to the constriction caused an increase in venous pressure, which resulted in bleeding.

A summary of the histories of two cases, showing marked difference in the degree of herniation through the esophageal hiatus, follows.

Case 1 (Fig. 5), a woman 55 years old, whose history previous to six months before consulting us contained no complaints suggestive of an upper abdominal lesion. At that time she began to regurgitate the last mouthful of food swallowed. At the onset, the regurgitation was intermittent, but for the last few weeks before consulting us the regurgitation followed nearly every meal and occurred at intervals varying from a few minutes to three or four hours after eating. The regurgitated food was described by the patient as "undigested and not mixed with stomach contents." Occasionally she was awakened during the night on account of nausea, which was relieved after expelling about a cupful of fluid.

Diaphragmatic hernia through the esophageal hiatus was not considered as a

probable cause for the complaint, although the history presented symptoms frequently complained of by patients with these herated on and a large uterine fibroid was removed. About five years before the gastro-intestinal examination she began to

Fig. 6.

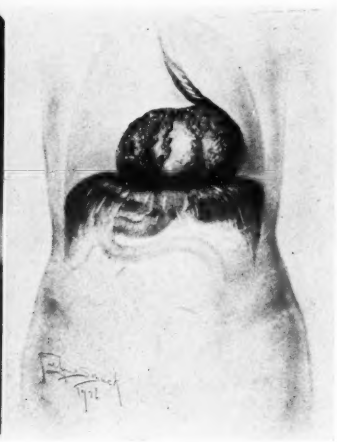


Fig. 7.

Fig. 6. Roentgenogram of Case 2, showing over half of the stomach herniated through the esophageal hiatus.
Fig. 7. Drawing illustrating the degree of herniation of the stomach as shown in Figure 6.

nias; neither was there any roentgenologic evidence of the hernia seen during the vertical fluoroscopic examination, notwithstanding the fact that a cardiospasm was suspected and special attention was paid to the lower end of the esophagus and cardiac end of the stomach. Only after viewing the roentgenograms taken in the supine position was the hernia recognized. It was fortunate that the hernia was visualized on the roentgenogram, for as a rule it is necessary to examine the patient in various oblique horizontal positions before such a hernia can be detected. Too much emphasis cannot be placed on the fact that the clinician must bear in mind the possibility of such a condition and the roentgenologist must be familiar with the clinical facts and use a technic necessary to demonstrate the hernia.

Case 2. The second patient was a 55-year-old woman whom the writer had known personally for many years. Her past history was essentially negative except that ten years before the roentgenologic examination of her stomach, she was oper-

have vomiting spells associated with a moderate amount of pain occurring within a quarter of an hour to one hour after eating, and the degree of distress was in proportion to the amount of food taken. Considerable palpitation and moderate dyspnea were also frequent complaints. The severity of the symptoms increased from year to year, until finally she was unable to sit through a moderate sized meal without leaving the table, walking about, and emptying her stomach by forcing herself to vomit. Roentgenologic examination (Fig. 6) demonstrated a large hernia of the stomach through the esophageal opening. Approximately half the stomach (Fig. 7) was herniated through the esophageal hiatus.

About a year ago this woman was suddenly stricken with severe pain in her chest which radiated down the arm, accompanied by palpitation and considerable dyspnea. A physician not aware of the presence of this large gastric hernia diagnosed the case as coronary thrombosis. The dyspnea, substernal distress, and pain radiating

down the arm lasted for five or six days, when the patient died. In the absence of electrocardiographic evidence and autopsy

through the foramen of Morgagni produces symptoms which are very confusing, suggesting decreased lung aeration, dullness at

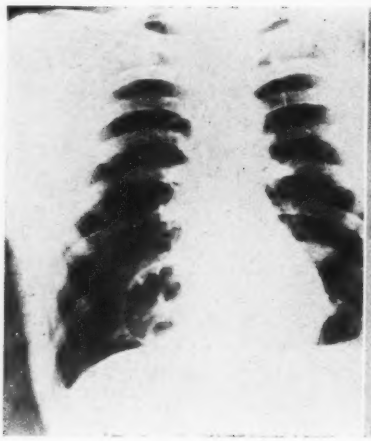


Fig. 8.

Fig. 9.

Fig. 8. Roentgenogram showing a right-sided hernia of the transverse colon through the foramen of Morgagni.
Fig. 9. Drawing illustrating the findings as exhibited in Figure 8.

findings, the question arises as to whether this large hernia might not have been the cause of death. Substernal pain radiating down the arm, dyspnea, and cyanosis have been described by various writers as caused by such a hernia, and on account of the size of this hernia relatively severe symptoms would be expected.

Diaphragmatic hernias through the pleuroperitoneal hiatus or foramen of Bochdalek and the dome of the diaphragm on account of the variation in size and location frequently involve other viscera than the stomach. The small and large bowel, spleen, and left lobe of the liver have been found herniated through these openings. The symptoms, therefore, are very indefinite, and the pathologic condition without roentgenologic findings would not be diagnosed clinically. The symptoms vary considerably with the viscera involved, and often because of symptoms suggesting cardiac and respiratory lesions the entire attention of the clinician is directed to the thorax. Herniation through the right side of the diaphragm other than the base, pleurisy, cyst of the liver, and bronchiectasis.

Many deaths in children attributed to thymus, congenital heart, atelectasis, and pneumonia are caused by a large diaphragmatic hernia.

Hernia through the foramen of Morgagni (Figs. 8 and 9) always involves the colon, and the clinical picture is that of partial or complete obstruction of the large bowel.

SUMMARY

1. The incidence of herniation of a portion of the cardiac end of the stomach through the esophageal hiatus is on the increase.

2. Herniation of a part or all of the cardiac end of the stomach is a definite clinical entity. The hernia produces suggestive symptoms and must be constantly borne in mind, both by the clinician and by the roentgenologist, as a possible cause of gastric complaints.

3. A hernia of the cardiac end of the stomach through the esophageal hiatus,

unless comparatively large, may exist for years without causing symptoms, and when demonstrated, other upper abdominal and mediastinal conditions capable of producing similar symptoms must be excluded before the hernia can be considered as the cause of the complaint for which the patient has sought relief.

4. Unless the roentgenologist has some reason to suspect the presence of a diaphragmatic hernia and examines the patient in a supine position, nearly all such hernias will remain undiagnosed.

REFERENCES

(1) HARRINGTON, S. W.: Diaphragmatic Hernia: Symptoms and Surgical Treatment in 60 Cases. Jour. Am. Med. Assn., Sept. 23, 1933, **101,** 907–914.
(2) HEDBLOM, C. A.: Diaphragmatic Hernia: Study of 378 Cases in which Operation was Performed Jour. Am. Med. Assn., Sept. 26, 1925, **85,** 947–953.
(3) RITVO, M.: Hernia of the Stomach through the Esophageal Orifice of the Diaphragm. Jour. Am. Med. Assn., Jan. 4, 1930, **94,** 15–21.
(4) HARRINGTON, S. W.: Diaphragmatic Hernia. Arch. Surg., January (pt. 2), 1928, **16,** 386–415.
(5) HEALY, T. R.: Symptoms Observed in 53 Cases of Non-traumatic Diaphragmatic Hernia. Am. Jour. Roentgenol. and Rad. Ther., March, 1925, **13,** 266–271.
(6) HUME, J. B.: Diaphragmatic Hernia. British Jour. Surg., April, 1932, **19,** 527–538.
(7) AKERLUND, A., OCHNELL, H., and KEY, E.: Diaphragmatic Hernia of the Esophageal Hiatus from an Anatomic and Roentgenologic Viewpoint. Acta Radiol. (Stockholm, in German), 1926, **6,** 3–22.
(8) HARRINGTON, S. W., referred to in "The Stomach and Duodenum," by George B. Eusterman and Donald C. Balfour, Saunders, 1935, page 693.
(9) BOCK, A. V., DULIN, J. W., and BROOKE, P. A.: Diaphragmatic Hernia and Secondary Anemia: Ten Cases. New England Jour. Med., Sept. 28, 1933, **209,** 615–625.

DISCUSSION

DR. ERIC J. RYAN (New York City): Dr. Dwyer is to be complimented on the thorough manner in which he has treated this subject. Herniation of the diaphragm is handled in a very sketchy manner in most of the standard text-books, and I think that this presentation, with its excellent résumé of the etiology, symptomatology, and diagnosis of this lesion goes far toward filling up the gaps in our knowledge.

Going back over the last 6,000 gastrointestinal examinations at St. Luke's Hospital, in New York, 19 cases of diaphragmatic hernia were reported—an incidence of 0.3 per cent.

As evidence that we are recording more of these cases in recent years than formerly, our incidence for 1932 was 0.17 per cent and for 1936, 0.44 per cent. This, I believe, is due to more careful study in at least two planes and to the fact that we have been stimulated to look more carefully for its presence.

Mention has been made of secondary anemia following bleeding due to congestion at the point of constriction. In our series of cases we have had two patients in whom ulceration occurred at this point and one case in which a massive gastric hemorrhage was the only symptom. This case was interesting from the point of view that the woman was "up in years." She was admitted with a diagnosis of gastric carcinoma—a large palpable mass. When she was x-rayed it was shown that most of the stomach, outside of the duodenum, was below the diaphragm; the large, palpable, nodular mass was a left scoliosis of the lumbar vertebræ with an advanced arthritis.

In only four of our cases were there symptoms that would suggest this lesion. All patients complained of being afraid to eat because of a sense of fullness and pressure in the chest. In addition, two of them —referred with a clinical diagnosis of carcinoma of the esophagus—gave a history of regurgitation if they partook of more than a certain amount of food. All complained of being afraid to eat.

All of our cases were herniations through the esophageal hiatus.

Careful examination in both the prone and erect positions, with the possibility of the presence of this lesion constantly in mind, will help us to recognize its presence in the majority of cases.

DR. R. R. NEWELL (San Francisco): There may be some hereditary factor in the tendency to develop hiatal hernias in advancing age. There is a Jewish family of eleven brothers and sisters of whom I have had opportunity to examine eight. All of those present hiatal hernias from 1 to 4 cm. in size. None produced symptoms.

These hernias are just one of the reasons

why we should always examine our gastro-intestinal patients supine and prone as well as standing.

DR. FRANCIS V. MARTIN (Michigan City, Ind.): I did not hear the first part of the paper, but, living as I do in a section where automobile accidents are perhaps more common than in most sections, I have seen two cases of ruptured diaphragm and in both of these I was able to foretell the condition by the clinical symptoms—by what I was able to hear and feel in the chest—by listening and palpation.

One could hear the bowel sounds away up in the chest where they should not be, and that, to me, in both those cases, allowed me to foretell the fact that we had a ruptured diaphragm with which to deal.

In one case the stomach, the spleen, the colon, and a large part of the small bowel were in the left chest, filling the whole chest cavity. The man died within a few hours. The other case was one in which a man was caught between two box cars. He was ill for months, and there was a lawsuit over the case. The judge appointed me to examine him. I found the greater part of the stomach in the chest cavity.

In both cases I was able to detect the probable condition by physical examination.

DR. ERNST A. MAY (East Orange, N. J.): In every gastro-intestinal examination we should look for a possible diaphragmatic hernia. Of the three methods which aid us in locating the presence of a diaphragmatic hernia, the Trendelenburg position is the most important one. Through the force of gravity alone the organs of the abdominal cavity, such as stomach, intestines, or omentum, may enter the chest through the defect in the diaphragm.

The second aid is the Mueller test. It consists of a forced inspiration which is suddenly stopped by allowing the patient to close the glottis while he is still trying to inhale. The diaphragm will descend to an extreme position and the hernia, through this low position as well as the increased negative pressure in the chest, will be larger.

The third is the Valsalva test. This method is not as useful as the Mueller test, but will aid in the differential diagnosis. After deep inspiration the patient closes the glottis and tries to make a forced expiration. In the abdominal type of breathing of the male, the increased abdominal pressure will force a part of the abdominal contents into the sac of the hernia above the diaphragm. The hernia, however, may disappear entirely, if, in the thoracic type of breathing of the female, the pressure in the thorax plus that of the diaphragm is greater than that of the abdomen.

As I have demonstrated in the case of bilateral diaphragmatic hernia which I reported in RADIOLOGY, April, 1933, fixation of the hernia in the chest by adhesions will change the results of these tests according to the amount of adhesions present.

DR. MAURICE F. DWYER (closing): I sincerely thank Dr. Ryan and the other gentlemen for their excellent discussion of this paper.

Throughout this paper I have tried to emphasize that hiatus hernias are not common, and when found, the physician must not consider these hernias as the cause of the symptoms for which the patient has sought relief until by careful examination he has excluded other conditions of the upper abdomen capable of producing symptoms closely simulating those of hiatus hernias. These hernias, however, are a definite clinical entity. Such a hernia may be the cause of the patient's complaint and should be thought of and recognized when present.

PRIMARY MALIGNANCY OF THE SMALL INTESTINE[1]

By EDWARD W. ROWE, M.D., *and* J. MARSHALL NEELY, M.D., *Lincoln, Nebraska*

INTRODUCTION

SINCE Lubarsch (15) described a malignant tumor of the small intestine in 1880, primary malignancy of the small bowel has been considered a rare occurrence, its diagnosis before operation unusual, and its roentgen interpretation so difficult that it was well nigh impossible. However, the increasing interest in the study, together with the stimulation of the advancements made in roentgenology, has revealed its more frequent occurrence, the possibility of pre-operative diagnosis and the lessening of the mortality rate. Thus have many dark fields in diagnosis yielded to routine accuracy in method and in determination of the correct interpretation of roentgen findings. It is not enough to wait for obstruction of the bowel or for the fatal signs of malignant disease. Medical service writes a proud chapter when a malignant tumor of the bowel is determined while there is yet time for palliation or for cure. Greater accuracy in diagnosis will result when there is better knowledge of neoplasms of the small bowel, of the clinical history, and especially of the normal and pathologic roentgen aspects. It is self-evident that greater care must be taken in the routine examination and special adaptation of the barium meal to the functions of the small bowel in health and disease.

STATISTICAL DATA

One of the most complete studies of tumors of the small intestine was published by Dr. T. S. Raiford (25), from the Surgical Department of Johns Hopkins Hospital, in 1932, who found 88 cases of tumors of the small intestine in a review of the records of 11,500 autopsies and 45,000 surgical specimens.

[1] Presented before the Radiological Society of North America at the Twenty-second Annual Meeting, at Cincinnati, Nov. 30–Dec. 4, 1936.

VARIATIONS AND LOCATIONS OF TUMORS OF THE SMALL INTESTINE (25)

Type	Duodenum	Jejunum	Ileum	Undetermined	Total
Carcinoma	7	4	3	2	16
Sarcoma	0	1	1	0	2
Lymphoblastoma	1	0	18	2	21
Adenoma	4	1	10	0	15
Myoma	0	1	2	0	3
Fibroma	1	0	3	0	4
Hemangioma	1	1	1	0	3
Chylangioma	0	1	0	0	1
Hematoma	0	0	1	1	2
Accessory Pancreatic Tissue	2	3	1	0	6
Argentaffin Tumor	1	1	5	0	7
Lipoma	3	1	3	0	7
Cyst	1	0	0	0	1
Total	21	14	48	5	88

In his review of the literature, Raiford (25) found 339 tumors of the small intestine. His own cases were distributed as follows:

All Tumors	Gastro-intestinal	986	
	Small intestine	88	(8.9%)
Benign Tumors	Gastro-intestinal	210	
	Small intestine	50	(23.8%)
Malignant Tumors	Gastro-intestinal	776	
	Small intestine	38	(4.9%)

In order of frequency, the terminal ileum leads in both benign and malignant tumors. The duodenum is next in susceptibility, and the jejunum is relatively free. Malignant tumors are usually large and single, involve the glands, and spread to the mesentery. Benign growths are smaller, often multiple, and usually polypoid.

Brill (1) reports 3,563 cases of malignant tumor of the intestine; 89, or 2.5 per cent, in the small intestine.

Doub (7), in 1936, reported 15 cases of primary malignancy of the small intestine, as follows:

(1)	Carcinoma of duodenum		9
(2)	Carcinoma of jejunum		3
(3)	Sarcoma of duodenum		1
(4)	Carcinoid carcinoma	(*a*) jejunum	1
		(*b*) cecum	1

Mateer and Hartman (17), in 1932, reported:

176,000 admissions to clinic (Detroit)
6 cases primary malignancy of intestine proven by autopsy

Forgue and Chavin (10), in 1915, reported:

88,031 autopsies, and cancer in 6,847 (8%)
642 cases (9%) intestine
603 " (94%) large bowel
39 " (6%) small bowel

Craig (5), in 1923, reported:

4,584 carcinomas of gastro-intestinal tract, and 36 of these were in the small intestine.

Nettrour (20), of the Mayo Clinic, 1936, stated in the Clinic's bulletin that carcinoma of the large bowel is eighty times as frequent as cancer of the small intestine. He found carcinoma of the small intestine most frequently in the jejunum.

PATHOLOGY

Primary neoplasms arising in the small intestine may be benign or malignant Benign tumors will be considered only briefly because this communication has to do with malignant neoplasms. Rankin and Newell (28) describe 35 cases of benign tumor of the small intestine, listing adenoma, myoma, fibroma, and lipoma. They state that benign tumors occur about half as frequently as carcinoma. Malignant tumors of the small intestine may be of epithelial or mesenchymal origin. Masson (16) has shown that the carcinoid tumor, which may be either benign or malignant, is of neurogenic origin.

The preceding tables show some discrepancy in the relative frequency of the various types of neoplasm. In Raiford's (25) series the lymphoblastoma group is most common. The series reported by Doub (7) shows adenocarcinoma most often. Adenocarcinoma is found in the jejunum more frequently than in the ileum or duodenum, according to Rankin and Mayo (27).

There are two types of carcinoma found in the small intestine: one develops from a pre-existing polyp, and the other simulates the ring-like, constricting carcinoma commonly found in the colon. In either of these two types obstruction is the rule, and metastasis occurs early. The mesenteric lymph nodes, peritoneum, liver, lung, long bones, and spinal dura are usually involved in the order named (27). In Craig's (5) report of 36 cases, metastasis occurred in 53 per cent. He found the prognosis worst in those cases in which the primary carcinoma was in the jejunum. Histologically, the most common type of carcinoma is the well-differentiated adenocarcinoma. Scirrhous or more undifferentiated types may be seen.

Five of the eight cases forming the basis for this paper are typical adenocarcinomas histologically, and all of them are located in the jejunum. All of these patients, including the one who is still living, show metastasis to the mesenteric lymph nodes. Distant metastasis did not occur in any case. The one remaining carcinoma is a colloid carcinoma which occurred in the first portion of the duodenum. Although the pathologist considered this as a primary carcinoma of the duodenum, at the time of autopsy, the fact that the pylorus was involved, with the histology, makes one feel that a primary carcinoma of the pylorus of the stomach is not entirely ruled out. This is particularly true when one remembers that colloid carcinoma of the stomach is not uncommon.

Carcinoid, or argentaffin tumors of the small intestine, do not occur in this small series of cases. Cooke (4), in an excellent review of cases, reports 11, three being malignant and eight benign. Intestinal obstruction was found in all those which were malignant and in one of the benign group. The tumor was found in the ileum in nine of these 11 cases. Though these tumors were formerly thought to be derived from epithelium, Masson (16) and others (4) have shown them to be neurogenic in origin. About 20 per cent of carcinoid tumors arising in the small bowel become malignant, metastasis being found most commonly in the regional lymph nodes and liver.

Sarcoma of the small intestine is rare, having occurred only twice in Raiford's series of 88 cases. The tumor may be either a fibrosarcoma or a myosarcoma: none occurred in this series.

The term "lymphoblastoma" as used here includes such pathologic entities as Hodgkin's disease, lymphosarcoma, and leukemia. Tumors of this type being prevalent in the fourth and fifth decades, may be expected at a slightly earlier age, and are more common in males than in females. In contrast to carcinoma, the small intestine is more commonly involved than the colon or stomach. The mesenteric lymph nodes are almost always involved though this involvement is simultaneous and not metastatic. In Raiford's (25) cases, lymphoblastoma is more common than carcinoma and is found most often in the ileum. Multiple tumors are frequently seen in Hodgkin's disease of the small intestine, though the single tumor is the rule in lymphosarcoma. Secondary inflammatory change often makes accurate histologic classification impossible in this group of tumors, and perforation of the intestinal wall, with resulting peritonitis, is not infrequent.

Two of the eight cases making up this group are lymphoblastomas. One of these (Case 7) is a typical abdominal Hodgkin's disease histologically and has been previously reported elsewhere (19). Multiple large polypoid tumors occurred in the jejunum and ileum and there was also multiple perforation of the bowel wall, with general peritonitis. Secondary inflammatory reaction made histologic classification difficult, but tissue remote from the areas of perforation revealed changes characteristic of Hodgkin's disease. The other lymphoblastoma was found in the terminal ileum and is probably a lymphosarcoma, although, as is indicated in the case report, there is a difference of opinion among various pathologists who have examined the material, one believing it to be Hodgkin's disease and another, lymphosarcoma.

CLINICAL HISTORY

As a rule, symptoms of malignant tumors of the small intestine are vague before the onset of complete obstruction. Pain, vomiting, increasing constipation, and rapid emaciation commonly occur. Doub has described a clinical picture for many types and locations, and such a detailed effort is commendable. In general, symptoms may be divided into those before and those after obstruction begins. All diagnosticians are fully informed of the signs of ileus and intussusception, but greater care in study of symptoms before either of these calamities occur is necessary, if a timely diagnosis is to be made.

Early appearance of visible peristalsis in any part of the abdomen and occult blood in the stool should make one think of tumor of the small intestine. In some cases, obstruction is so late that anemia, loss of weight and cachexia are present to a greater or lesser degree. There is a group of symptoms arising in the greater number of cases, in which the complaints point to the region involved and constitutional changes indicate profound illness. A lesser group occurs in which the outstanding symptoms are constitutional and the regional manifestations are obscure. Gradual occlusion by the growth gives rather characteristic symptoms of belching, bloating, and a feeling of heaviness in the abdomen. The patient is below par, fatigues easily, his weight decreases, and the symptoms of obstruction precipitate, to remit again or to remain for the final stage of total occlusion. Slight colicky pain in the abdomen, waves of peristalsis, nausea, and later, vomiting should, with a palpable tumor, localize the region involved. Bowel movements may be normal at first and then become infrequent, or the stool may become scanty and tenesmus be troublesome. As a rule, the higher the tumor the more violent the symptoms and the earlier vomiting occurs.

The clinical findings are usually divided into (1) those of the duodenum, and (2) those of the jejunum and ileum. The latter are sufficiently alike to be described together. The malignant tumors of the jejunum and ileum have fewer localizing symptoms and signs. The length of the prodromal signs varies greatly. Some-

times four to six months may intervene before obstructive signs appear. Usually the onset is sudden.

In carcinoma of the duodenum, the first and third portions may be grouped together. (1) Pain appears—dull, dragging, not related to meals, paroxysmal, seldom referred, diffuse in epigastrium, and more intense in the region involved. (2) Nausea and vomiting are common complaints, occurring later than pain. Hematemesis is common. (3) Loss of weight and cachexia are marked as in the other gastro-intestinal malignant tumors. (4) Constipation is generally the rule, but diarrhea may alternate. (5) Melena. Occult blood is usual, but black or tarry stools may be seen.

The physical findings of the first and third portions of the duodenum are (1) tenderness, which may be slight or exquisite, and which is most intense over the tumor mass. Rigidity may or may not be present. (2) Mass may be felt if large enough. It is usually resistant and fixed. (3) Ascites—if extensive—points to portal obstruction and metastasis. (4) Gastric analysis reveals low, or the absence of, HCl. (5) Blood is usually demonstrated as a gross or occult finding in the stool. (6) The blood picture shows profound anemia, in advanced stages. The white count is not elevated. (7) Pulse and temperature are not remarkable, as in anemia.

The clinical findings of the second portion of the duodenum are the same, with additional features: (1) Pain is more acute as obstruction of the pancreatic duct and biliary passage becomes marked. Back pressure and the chemical action of retained pancreatic juice cause great distress. (2) Jaundice appears early and is usually painless: it may progress rapidly without remission. Intense itching may occur. (3) The stools become clay-colored, due to the absence of bile.

The symptoms and findings of malignant tumors of the jejunum and ileum are classed together because they are similar except for the localizing value of a tumor mass, if it can be palpated. When obstructive symptoms are present in the younger ages, intussusception is usually diagnosed; in the older, it is obstruction by slow encroachment upon the lumen of the gut, and the constitutional symptoms predominate. Recognition of the diagnostic criteria earlier than obstruction of intussusception offers the most hope. Resection of the growth and possibly roentgen therapy must be early if a life is to be saved.

ROENTGEN DIAGNOSIS

To an increasing number of the medical profession it appears that careful roentgen observations of the small intestine offer the same exactness in diagnosis as in the pylorus, the cap, or the cecum. In a review of many reports, it appears that malignant tumors of the small intestine have not been recognized often by the roentgen examination. Usually an exploratory operation has revealed the pathology.

Intestinal obstruction, intussusception, acute abdomen, and exploration of a suspected tumor mass are the common preoperative reasons given for the operation. In most cases, the roentgen examination, if employed, has consisted of preliminary roentgenography or a routine gastro-intestinal examination, not timed for the special observation of the small intestine. Recent reports indicate that there is an awakening interest in the early diagnosis of this condition.

I quote from "Roentgenographic Technic," by Rhinehart (29):

> "Except the duodenum and the terminal part of the ileum, little attention is paid to the small intestine in the usual roentgenologic examination of the gastro-intestinal tract. Carman says that 'practically the sole abnormality of the jejunum (and upper ileum) showing roentgenologic signs is obstruction.' Mills attempted to draw conclusions from abnormalities in the appearance of the small intestine which he believed were due to derangements in the tension of the gastro-intestinal tract."

Morse and Cole (18), in 1926, read before this Society a most complete study of the anatomy of the normal small intestine as observed roentgenographically. They believed that a procedure of roentgenographic examination could be devised which would enable one: (1) To recognize the roentgenographic appearance of the normal small intestine, together with those variations in appearance which come within so-called normal limits; (2) to study the motor phenomena of the small intestine; (3) to recognize and identify pathologic conditions involving the small intestine and functional derangements of the small intestine.

Their study of the contour, caliber, and the groupings of the small intestines is classic and worthy of the foundation for all reports upon this portion of the digestive tract. Very definitely the coils may be found in certain portions of the abdomen if observed at the proper time, exactly as they described them; and when the patterns or the positions vary, some explanation must be offered to explain the variance.

Again, in 1935, Dr. Cole and Dr. Pound (3) offered a valuable contribution to the knowledge of the small intestine, discussing, (1) the normal and (2) the interference of inflammatory lesions with the motor phenomena.

The work of Pendergrass and his coworkers (23) has been most thorough. They have called attention to the variations in the emptying time of the stomach and small intestinal patterns under a variety of conditions which may be imposed on the gastro-intestinal tract, by lesions not primarily involving the tract itself, by drugs, and by operation. He points out the groupings of the small intestines, their change in contour, and their individual patterns, how they appear normally with different types of contrast medium. He introduces a terminology—segmental contractions, feathery snow flakes, bolus, herring bone, coin shapes, piled coins, trabeculation patterns, degrees of motility, etc., and helps to give them definite meanings. To many, the small bowel simply lies within the abdominal cavity, beginning at the pylorus and terminating at the cecum. In between lies a no-man's-land of such protean patterns that it is hardly adaptable to scientific description.

Similar illuminating studies on the action of the small intestine have been made by Etienne Piot (24), in France, and by Hans Pansdorf (22), in Germany. There seems to be agreement now on Forssell's mucosal formations and rapid changes in patterns, Ludwig's pendulum movements and peristaltic waves, Cannon's segmentation and rosary-like formations, that movements in the small bowel are more rapid in the upper tract and quieter in the distal, as in the colon, that there is no rhythmic discharge of the ileocecal valve, and that the valve seldom if ever protrudes into the cecum, that antiperistalsis has rarely if ever been seen, and that palpation promotes evacuation of intestinal loops.

It has been found that certain conditions and drugs must be understood or they complicate the picture. Hyperthyroidism causes increased motility. Myxedema causes slowness and slowed evacuation. Inanition delays evacuation. Loss of consciousness stops all movements. Atropine slows all movements. Fat delays grosser movements, and achylia increases motility.

The roentgen examination of the small intestine, properly carried out, is unfortunately a time-consuming and relatively expensive procedure. In properly selected cases, it will disclose the solution to an otherwise puzzling problem. Clinicians are recognizing more and more the value of a careful examination in obscure lesions of the gastro-intestinal tract. Positive findings depend on the stage of the disease. They are not always infallible, and because of the lateness of an opportunity to study the usual case, the findings may not be very determinate. Even positive findings may not be pathognomonic: neither do negative findings exclude the possibility of a tumor. There must be careful and intelligent correlation of the clinical find-

ings with the objectives of the roentgen examination.

The duodenum is best seen in the first half-hour after the ingestion of the meal. Its behavior in this period should indicate if a more detailed examination than usual is desirable. Study of the small intestine should begin when the stomach first begins to empty. After the examination of the stomach further observation of the small intestine should be made at half-hour intervals—at six hours for ileum, at nine hours for the ileal stasis, and again at twenty-four hours for occasional ileal stasis. Ileal stasis, or gas, in the small intestine always need explanation. A barium enema should be employed not only for colon but also for a complete ileal study. Sometimes the contrast enema is an advantage.

In the duodenum may be found: obstruction; unusual motility; dilatation; destruction of pattern; filling defects; narrow, irregular passage if produced by encroachment on the lumen, or a narrow canalization. In the jejunum may be revealed the same findings plus the tumor itself. The outlines of a tumor may be seen on the preliminary films and confirmed with the barium meal. In either, the assumed patterns may indicate the pathology. The true filling defect, which is an irregular outline of a protruding growth into the lumen of the bowel, is as pathognomonic as in the stomach wall. Distal ends have appeared bulbous, revealing the outlines of growth as well as the obstruction.

CONCLUSIONS

In view of the rare occurrence of tumors of the small intestine, which have been diagnosed as such by roentgen observation or even after complete clinical study, a task has been set for the roentgenologist. Cases are usually diagnosed by the surgeon as intussusception or obstruction of the bowel, and the true pathology has been revealed either at operation or at biopsy or postmortem. The roentgen findings of the normal small intestine vary much in health with each individual and with different types of media used in the visualization of the contour and mucosa. Abnormal findings from drugs or from extrinsic pathology are so commonly met that their presence must be anticipated. Painstaking technic especially adapted to the study of the small intestine must be carried out, if the true pathology is to be determined in time for relief.

An increasing number of primary malignant tumors of the small intestine have been recognized by roentgen examination before operation. Two of the cases whose history follows have been treated, one surgically and one by roentgen therapy, and are alive to-day. It is just as certain in this field as in others, where the criteria of diagnosis are well known, that findings of high accuracy will in time appear in our literature and be accepted in the routine day's work.

TABLE I

	Lancaster County Laboratory	Bryan Memorial Hospital	Lincoln General Hospital	Lincoln Clinic
No. Patients	...	14,418	30,032	28,260
Autopsies	1,191	....	....	....
Tumors of Gastro-intestinal Tract	91	....	....	112
Malignant Tumors of Gastro-intestinal Tract	74	....	....	107
Primary Malignant Tumors of Small Intestine	8	3	5	4

Location of Tumors:		Living Patients:	
Duodenum	1	Resection jejunum	1
Jejunum	5	Roentgen Therapy of Ileum	1
Ileum	2		
Total	8	Total	2

CASE REPORTS

Case 1. Mrs. E. E. L.; examined Aug. 4, 1928; referred by Dr. J. S. W. and Dr. H. J. L.; aged 42 years. Complains of gas accumulation, bitter taste and nausea, which began a year before and are growing worse. Spells of vomiting large quantities of green bile or food, sometimes amounting to a quart. There are short respites between severe attacks. Pain is frequent in upper abdomen, radiating to the right

shoulder blade; relieved by vomiting. The appetite has been fair until recently, but is becoming poor. When gas is absent, she is hungry. Patient is constipated.

Loss of weight is questionable; color fair; abdomen soft but generally tender. Liver tender and margin felt one inch below the costal arch. No peristaltic waves are seen; no masses palpated; no gas distention. Temperature, 99 degrees; pulse, 100; blood pressure 70/180.

Blood: hemoglobin, 28 per cent; red blood cells, 2,500,000; white blood cells, 5,800.

Four stools were examined: occult blood was found in three; no gross pus, bile or blood.

Roentgen Examination.—"The duodenum was full and dilated, giving one the impression of obstruction lower in the small bowel. At the end of 24 hours there were traces of barium in the stomach."—(Dr. R. L. S.)

The patient refused surgery. Ten months later, she died.

Protocol.—"Stomach dilated to three times its normal size. Lining mucosa folds are widely separated. Scar on greater curvature. Duodenum greatly dilated. Jejunum for nine inches gives the appearance of a second stomach. Distal to the out-pouching there is an irregular lumen. On section, the ring is found to occupy about one inch of the intestinal wall. It is rather friable, but cuts with increased resistance. This growth occurs in the first foot of the jejunum. The remainder of the small and large intestine is essentially negative.

"Cause of death: Carcinoma of jejunum with metastasis in mesenteric glands and liver."

Pathologist's Report.—"Tumor: papillary adenocarcinoma of jejunum."

Histopathology.—"A more or less normal mucosa covers the area. The gut wall is markedly thickened by the tumor. The muscle layers are intact but widely separated by loose papillary carcinoma. That on the serosa is much more compact."

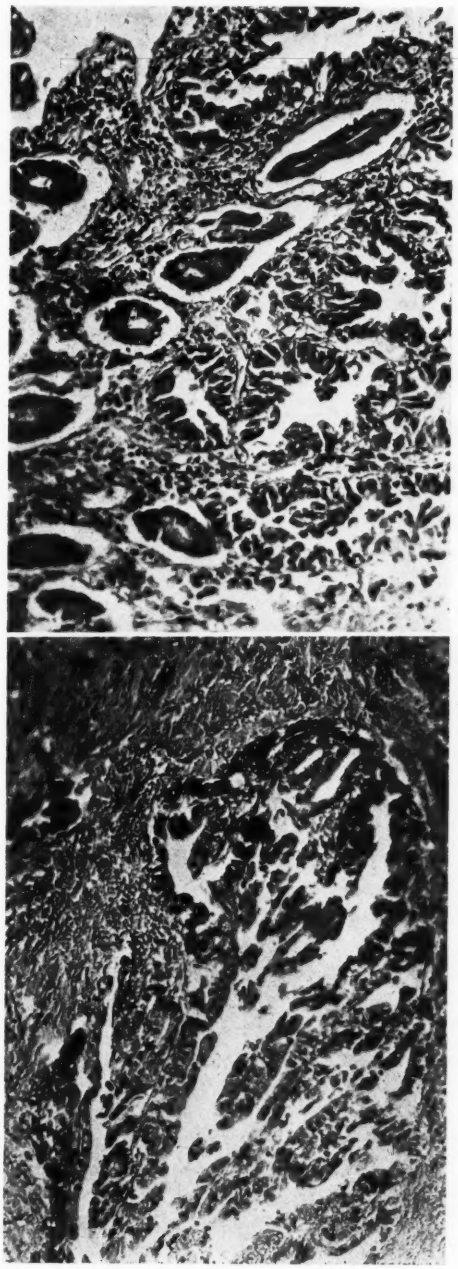


Fig. 1 (*above*) and Fig. 2 (*below*). Case 1. Papillary adenocarcinoma of jejunum.

Case 2. Mrs. Jennie H.; examined April 1, 1929; referred by Dr. J. S. W. and Dr. H. J. L.; aged 52 years; widow; housewife.

History.—Menopause three years previously. For 17 years she has been having attacks similar to the present, overwork or anxiety precipitating them. The present trouble began three months before examination with epigastric distress after meals and at bedtime.

Present Symptoms.—(1) Pain in epigastrium, not radiating. Intervals of relief. Relieved by food and soda. Lately, some griping pain in abdomen below navel. (2) Gas accumulation above navel (present for 16 years). Little belching. (3) Ten-pound loss of weight in a year.

Examination.—Pulse 108; blood pressure, 172/90; temperature, 98°. Slight resistance in the epigastrium; no tenderness. Pelvis—multiple fibroids. Urine: specific gravity, 1.015; acid; albumin, 0; sugar, 0; microscopic examination, negative. Blood: hemoglobin, 70 per cent; red blood cells, 4,350,000; white blood cells, 4,800. Basal metabolism: + 16.

Roentgen Examination.—"Chronic duodenal ulcer."

Operation.—April 15, 1929, the gall bladder and pylorus were explored; found scar and adhesions of duodenal ulcer. Did a subtotal hysterectomy and appendectomy.

Death.—April 17, 1929. Cause, "Chronic nephritis. Contributory: carcinoma of jejunum, thyroiditis and myocarditis."

Protocol.—"At the beginning of the duodenum, just beyond the pylorus at the posterior aspect, there is a healed ulcer scar. In the mid-portion of the jejunum there is a fibrinous band about the bowel, three-quarters of an inch in diameter. It is firm and on opening the bowel it appears to form a definite annular constriction. It gives an impression of an annular carcinoma. The mesenteric glands are enlarged and cut with resistance."

Fig. 3.

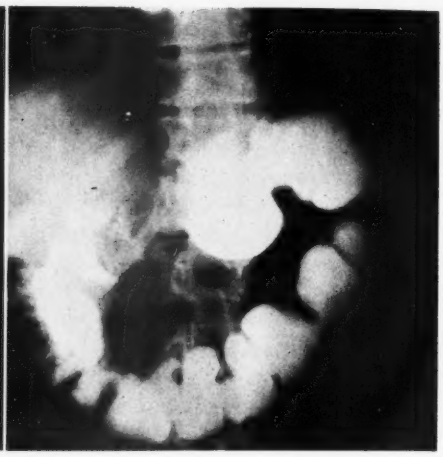


Fig. 4.

Figs. 3 and 4. Case 5. Filling defect of the duodenum. Diagnosis: carcinoma of the duodenum.

Histopathology.—"Small intestine—jejunum shows infiltration of the submucosa and muscularis coats of carcinoma cells, some taking on glandular formation. Some areas of villi are being infiltrated by a carcinomatous growth. Also a considerable amount of round-cell infiltration through the section. . . . . Adenocarcinoma of jejunum."

Case 3. Mrs. Emma McV.; examined Oct. 23, 1934; referred by Dr. F. S. F.; aged 46 years; menopause three months previously. *Chief Complaints.*—Nervousness, poor appetite, loss of weight, shortness of breath, and pain in the lumbar area. Six months before examination she had been operated on in the University Hospi-

tal for a ventral hernia and a mass which was considered malignant and inoperable was found in the abdomen. The ventral hernia was closed. Because of an internal hemorrhage she had a stormy convalescence. Spinal anesthesia was used. She recovered, but complained of pain in the lower legs accompanied by anesthesia, which she attributed to the spinal block.

Examination.—Below the umbilicus in the mid-line and extending into the left side was a hard, fixed mass, the size of an orange, tender on pressure. Urine examination showed a trace of albumin; microscopic pus. Blood: hemoglobin, 24 per cent; red blood cells 1,800,000; white blood cells 7,800.

Diagnosis.—General carcinosis of unknown origin.

Dyspnea and weakness grew worse. There was no vomiting until the last day. She died five weeks after entering the hospital. There was no record of any roentgen examination.

Protocol.—"In the small intestine about three feet from the pyloric ring at what appears to be the jejunal area or near the proximal end of the ileum is a large mass two inches in diameter. Upon section of the bowel at this point, the mass is found encircling the bowel and has the appearance of circular, infiltrating, primary malignant tumor. The tumor extends into the lumen of the bowel and apparently has caused almost complete obstruction. The remainder of the small intestine shows nothing remarkable except some isolated nodular tumors. The mesenteric lymph nodes are enlarged and show metastatic growths."

Pathological Report.—"Adenocarcinoma of jejunum."

Case 4. Mrs. Pauline R.; examined May 24, 1932; referred by Dr. J. S. W. and Dr. O. E. L.; aged 26 years. Brought to the hospital suffering from acute localized pain in the right lower quadrant. Pulse 90. Temperature 99 degrees. A palpable tumor with moderate rigidity and tenderness. There was nausea, and vomiting had occurred. The pain was severe. The bowels had been evacuated by enema. White blood count 6,500. Diagnosis: Acute appendicitis, with reservations.

Fig. 5. Case 5. Colloid carcinoma primary in the duodenum.

At operation the appendix was found slightly injected, and was removed. The surgeon doubted its connection with the clinical history. The ileum was distended, and proximal to the ileocecal valve was a firm, nodular tumor involving the bowel wall and surrounding lymph nodes. This was thought by the surgeon to be malignant. The biopsy was reported by one pathologist as "lymphosarcoma"; by another as "Hodgkin's." Final diagnosis: "Ileus from lymphosarcoma involving the ileum."

Progress was slow and stormy. Gas distention, loss of appetite, and pain. Twelve days later roentgen therapy was begun. Recovery followed promptly.

Patient returned one year and ten months later, with complaints of slight pain and a barely palpable tumor in the right iliac fossa. A second series was administered, with complete relief.

The patient is now, two and a half years later, in excellent health. Blood picture normal.

"Malignant mass, right upper quadrant of abdomen, probably a newgrowth from the pylorus."

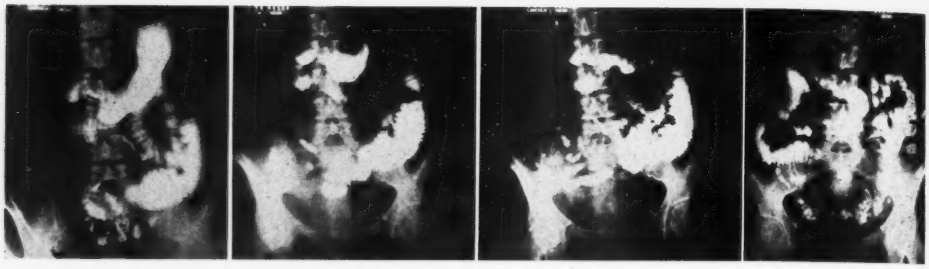


Fig. 6-*A*, 30 min. Fig. 6-*B*, 3 hr. Fig. 6-*C*, 5 hr. Fig. 6-*D*, 24 hr.
Figs. 6-*A*, 6-*B*, 6-*C*, and 6-*D*. Case 6. Shows filling defect of the jejunum, as well as stenosis. Ballottement of tumor on fluoroscopy. Carcinoma of jejunum.

Case 5. William A. S.; examined March 1, 1934; referred by Dr. D. C. H.; aged 60 years; married: architect by occupation. The patient is bedfast, weak, pale; hands and feet are puffy; cyanosis; mentally clear and hopeful. Ten months previously occurred onset of "stomach trouble." Food would "go right through," though previously he had been constipated. Weakness came on gradually. Two weeks previous to examination he took to his bed. He had no pain or bleeding except from small piles. He had never been seriously ill; had had no injuries; his general health prior to his present acute condition had been fair. Abdomen soft and relaxed; palpable line and nodular; no marked tenderness; no resistance.

Examination.—Blood: hemoglobin, 20 per cent; red blood cells, 1,840,000; white blood cells, 10,800; erythrocytes very pale. Anisocytosis 1 +; poikilocytes 1 +; polychromatophilia 1 +; normoblasts 0; Wassermann negative. Impression: secondary anemia.

Roentgen Examination.—"The cap and first portion of the duodenum are irregular in outline, firmly fixed and embedded in a mass apparently arising in the pyloric end of the stomach. The duodenum and jejunum are fixed in the mass which extends down into the right side of the abdomen. It also contacts with the wall of the hepatic flexure of the colon." Interpretation:

Protocol.—"The stomach is dilated, containing much fluid and gas. The mass, which is large, invades the duodenum and pushes the colon and gall bladder to the right. On opening the duodenum it is found to contain papillary areas which are typically malignant."

The pathologist on section of the tissue reported: "Colloid carcinoma, primary in duodenum."

Case 6. Mrs. Mary M.; examined March 3, 1936; referred by Dr. G. H. M.; aged 61 years; widow; native of Germany.

History.—Complains of nausea, vomiting, and cramp-like pain in the left upper abdomen. Nine months previously she suffered some pain, with vomiting. Nine days prior to examination, pain began again and has occurred daily since. Six weeks previously, gastro-intestinal barium meal examination was made elsewhere (Dr. F., Omaha) and reported: "Normal gastro-intestinal tract." Lately, she has noticed a bulging of the left upper abdomen, accompained by pain. Appendectomy and suspension of uterus in 1917. Surgical drainage of gall bladder in 1927.

Examination.—Tumor in abdomen. Intestines can be seen to bulge and disappear, accompanied by pain and nausea. Fractional analysis shows marked hyperchlohydria; no blood. Cholecystographic; examination shows normal gall-bladder function. Blood: hemoglobin, 65 per cent;

red blood cells, 4,110,000; white blood cells, 8,400. Electrocardiograph suggests myocardial damage.

X-ray Report.—"Stomach steerhorn type hypertonic, normal rugæ and contour. Six-hour stasis 1+. Small intestine: cap full and complete, duodenal curve regular but dilated beyond to distal portion of jejunum, demonstrated at three hours, five hours, and 24 hours. At end of dilated area there is a definite filling defect which is characteristic of carcinoma of jejunum. It is palpable. Filling defect seen on fluoroscopic and roentgenographic examinations—not just stenosis. Almost complete obstruction."

Surgical Report.—Carcinoma of jejunum. White, stenosing mass in jejunum 2 feet below the ligament of Treitz. Lymph node the size of walnut in mesentery. Chronic obstruction of jejunum. Resection of mass in jejunum and associated enlarged nodes. Side-to-side anastomosis of jejunal ends."

Recovery; good condition at present time (July 10, 1936). This is the only case diagnosed positively before operation, and also the second of eight reported.

Case 7. Mr. C. A. B.; aged 65 years; glass blower by occupation.

Abstract of report in "Nebraska State Medical Journal," September, 1934: "When admitted to the Lincoln General Hospital the patient was complaining of severe intermittent pain in the lower abdomen, gaseous eructations, and loss of appetite. Family history reveals the fact that the father died of lower abdominal tumor of unknown origin. In 1896 the patient had severe pulmonary hemorrhage, and in 1905 a severe gastric upset of some kind. Vague lower abdominal distress existed for three months. One month before admission he had a chill lasting 45 minutes and pain in the right kidney region. Three weeks later he was cystoscoped and the right ureter dilated. This was followed by a chill and pain in the right lumbar region. For two weeks before admission the patient noticed intermittent, rhythmic lower abdominal pain.

"A barium meal series two days following admission revealed nothing remarkable. Urologist diagnosed right ureteral obstruction and explained the gastro-intestinal

Fig. 7. Case 8. Carcinoma of jejunum.

symptoms as reflex. The right kidney was removed May 1, 1933. Examination of kidney disclosed pyonephrosis with obstruction at the uretero-pelvic junction and what appeared to be inflammatory tissue. Patient left hospital in 18 days, after stormy post-operative course with intermittent lower abdominal pain.

"Patient was re-admitted May 29, 1933, with severe pain in the left flank, which began suddenly on the day of admittance. Temperature 100 degrees, rose to 101 degrees. Report on preliminary radiographs is as follows: 'Gas in splenic and hepatic flexure. Also gas is seen in the small bowel, which indicates an intestinal obstruction. Barium enema shows spasticity of the sigmoid region with definite dilatation of the splenic flexure and some dilatation of the transverse colon. Gas remains in small bowel, as seen in the flat film. There is no evidence of filling defect in the colon.'

"On May 29 exploratory examination was done and generalized peritonitis found. Several polypoid tumors of small intestine were encountered, one which was obstructing and through which there had been perforation. It was resected. Patient became worse and died May 30, 1933.

"*Report of Necropsy:* On following the ileum proximally from the cecum it is found to extend deep into the pelvis at

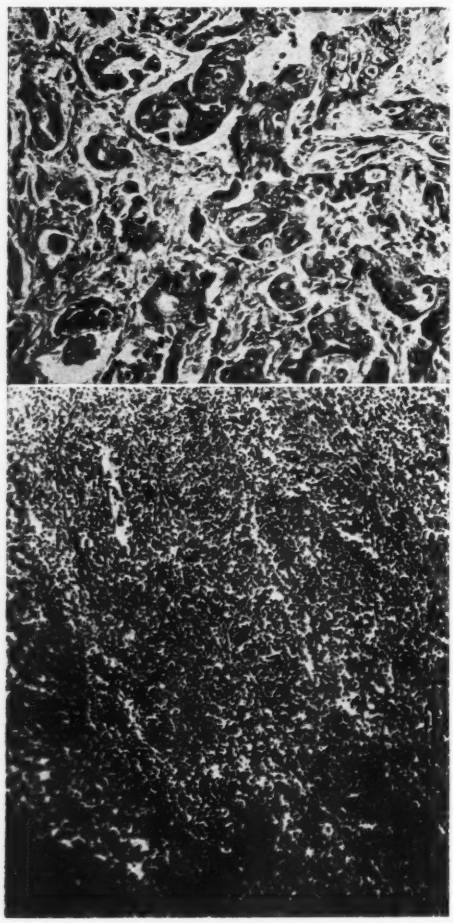


Fig. 8 (*above*). Case 2. Photomicrograph of tissue, adenocarcinoma of jejunum.
Fig. 9 (*below*). Case 7. Photomicrograph of tissue, Hodgkin's of the small intestine. One of two cases involving the ileum. (Case 4, similar, alive and well after roentgen therapy.)

which place it is adherent to the urinary bladder by both fibrous and fibrinous adhesions. Just proximal to this point of attachment there is a perforation of the bowel about which there is a thick layer of fibrinous exudate. No leak. The entire intestinal tract is markedly distended and mesenteric glands throughout are markedly enlarged and are firm to palpation.

"*Anatomical Lesions:* Multiple tumors of small intestine (lymphoblastoma) benign prostatic hypertrophy, general fibrinous peritonitis, multiple perforation of the small intestine, post-operative intestinal resection and nephrectomy, etc.

"*Cause of Death:* Generalized peritonitis and ruptured intestine.

"*Microscopic:* Section of tumors occurring in the small intestine show them to be made up of varying sized and shaped mononuclear cells. The tissue is literally loaded with both mononuclear and multinucleated giant cells. Many types of cells are emeshed in a very markedly hyperplastic reticulo-endothelial network. . . . . The tumor apparently started in the mucosa and submucosa and has deeply infiltrated the muscularis almost to the serosa. Ulceration of overlying mucosa is seen. Sections of mesenteric nodes, and of tissue from the region of the right ureteropelvic junction show typical Hodgkin's tissue."

SUMMARY AND CONCLUSION

1. A somewhat unusual case of abdominal Hodgkin's disease has been presented.

2. There is nothing constant in either subjective or objective findings which made the clinical diagnosis of Hodgkin's disease positive.

3. The fact that Hodgkin's disease, like lymphosarcoma, may involve the gastrointestinal tract further supports the idea held by many, that these two pathological entities are probably slightly different manifestations of the same disease.

Case 8. Mrs. Nellie H.; aged 67; housewife, referred by Dr. G. H. W., Lincoln General Hospital, October 18, 1932, for examination. The complaints were (1) vomiting, (2) constipation, (3) nervousness.

History.—The onset occurred five months previously when the woman suddenly began to vomit. Nausea and vomiting have gradually been getting worse, until now she vomits on the least cause. Constipation has been growing worse for five

months, going three days without a bowel movement. The appetite has been generally good until a few days previous to examination. She has had no relevant diseases.

Physical Examination revealed an old, dehydrated woman with enlarged heart and râles in the lungs. The abdomen was distended. In the upper abdomen was the outline of a "tumor," which disappeared after she vomited two quarts of bile and duodenal contents: has remained empty since fluids were discontinued.

Roentgen Examination (Oct. 20, 1932).—"Definite obstruction 15 or 18 in. distal to the duodenal cap, probably at the terminal duodenum. This stenosis, which is practically complete, has produced considerable dilatation above it. Whether it is due to malignancy or to an old inflammatory affair, or adhesions, I can't say." (Dr. R. L. S.)

Consultation with Internist.—Impression: "Obstructing lesion in upper small intestine, probably jejunal. Character of lesion not evident. Primary carcinoma here is rare."—(Dr. G. W. C.)

Exploratory Operation (Oct. 22, 1932).—"Dilated jejunum and an obstructing tumor were found. Resection of gut and end-to-end anastomosis. Bad risk."—(Dr. H. H. E.)

Death occurred Oct. 26, 1932, four days after operation.

Pathological Report.—"A section of the jejunum 10 cm. long. There is a constriction in the middle with white collar-like strip about one centimeter wide extending three-fourths of the way around the circumference; beneath this a mass is palpable. A hard thickening of intestinal wall. The mass obstructs the lumen almost entirely. On section, this mass leaves a firm, smooth, white surface.

"Microscopic Findings: The structure is that of a hyperplastic acinous glandular tissue in which the individual alveoli are poorly formed. The epithelial cells are large, contain considerable cytoplasm, and have a large palely stained nuclei, with well-defined nuclear membranes and prominent chromatin granules and networks showing all the characteristics of rapid growth.

"Diagnosis: Adenocarcinoma of the jejunum."

BIBLIOGRAPHY

(1) BRILL, N. E.: Am. Jour. Med. Sci., 1904, **128,** 824.

(2) BUSCHE, H. J.: Roentgen Diagnosis of High Seated Sarcoma of Small Intestine. (Roentgenpraxis, March, 1934, **6,** 141–145) Yearbook of Radiology, 1934, p. 210.

(3) COLE, LEWIS GREGORY, and POUND, ROBERT E.: Small Intestine—Inflammatory Lesions and Accompanying Reactions. RADIOLOGY, September, 1936, **27,** 330–338.

(4) COOKE, H. H.: Carcinoid Tumors of the Small Intestine. Arch. Surg., April, 1931, **22,** 586–597.

(5) CRAIG, W. MCKANE and MACCARTY, W. C.: Involvement of Lymph Glands in Cancer of Cecum. Ann. Surg., June, 1923, **77,** 698–710.

(6) DARDINSKI, VINCENT J.: Primary Carcinoma of the Duodenum. Am. Jour. Path., March, 1934, **10,** 313–318.

(7) DOUB, H. P., and JONES, H. C.: Primary Malignant Tumors of the Small Intestines. RADIOLOGY, February, 1936, **26,** 209–220.

(8) EGER, S. A.: Primary Malignant Disease of the Duodenum. Arch. Surg., December, 1933, 1087–1108, Yearbook of Radiology, 1934, p. 206.

(9) EWING, JAMES: Cancer of Duodenum, Jejunum, and Ileum. Neoplastic Diseases, 1928, pp. 704, 705.

(10) FORGUE and CHAVIN: Rev. de chir., 1915, **44,** 470.

(11) GOLDEN, ROSS: The Roentgen-ray Examination of the Digestive Tract: Duodenum, Jejunum, and Ileum. Diagnostic Roentgenology, 1936, p. 294.

(12) HARTMAN, HOWARD R.: Lesions of the Small Bowel Other Than Peptic Ulcer. Med. Clin. No. Am., September, 1935, **19,** 365–382.

(13) HOHENNER, KARL: Early Diagnosis of Carcinoma of Small Intestine. Röntgenopraxis, October, 1934, **6,** 677–679, Yearbook of Radiology, 1935, p. 226.

(14) HUMPHREYS, ELEANOR M.: Carcinoid Tumors of the Small Intestine: Report of Three Cases with Metastases. Am. Jour. Cancer, December, 1934, **22,** 765–775.

(15) LUBARSCH, O.: Arch. f. path. Anat., 1888, **111,** 280.

(16) MASSON, P.: Carcinoids (Argentaffin-cell Tumors) and Nerve Hyperplasia of Appendicular Mucosa. Am. Jour. Path., May, 1928, **4,** 181–212.

(17) MATEER, J. G., and HARTMAN, F. W.: Primary Carcinoma of the Duodenum. Jour. Am. Med. Assn., Nov. 26, 1932, **99,** 1853–1885.

(18) MORSE, R. W., and COLE, LEWIS GREGORY: The Anatomy of the Normal Small Intestine as Observed Roentgenographically. RADIOLOGY, February, 1927, **8,** 149–153.

(19) NEELY, J. M., JR.: Abdominal Hodgkin's Disease. Nebr. St. Med. Jour., September, 1934, **19,** 336–339.

(20) NETTROUR, W. S.: Carcinoma of the Jejunum: Report of Case. Proc. Mayo Clinic, June 3, 1936, **11,** 356–360.

(21) OCHSNER, ALTON: Malignant Tumors of the Small Intestine. Nelson's Loose Leaf Living Surgery, Vol. V, pp. 288–291.

(22) PANSDORF, HANS: Experimental Studies in Roentgenology of the Small Intestine. Ergbn. d. med. Strahlenforschung, Vol. 5: Yearbook of Radiology, 1932, p. 160.

(23) PENDERGRASS, E. P., RAVDIN, V. S., JOHNSTON, C. G., and HODES, P. J.: Studies of the Small Intestine. RADIOLOGY, June, 1936, **26,** 651–662.
(24) PIOT, ETIENNE: Radiologic Study of the Small Intestine. Jour. de radiol. et d'électrol, April, 1932, **16,** 175–177: Yearbook of Radiology, 1932, p. 162.
(25) RAIFORD, T. S.: Tumors of the Small Intestine: Their Diagnosis, with Special Reference to the X-ray Appearance. RADIOLOGY, February, 1931, **16,** 253–270.
(26) Idem: Tumors of the Small Intestine. Arch. Surg., July, 1932, **25,** 122, and August, 1932, **25,** 321.
(27) RANKIN, F. W., and MAYO, CHARLES, JR.: Carcinoma of the Small Bowel. Surg., Gynec. and Obst., June, 1930, **50,** 939–947.
(28) RANKIN, F. W., and NEWELL, C. E.: Benign Tumors of the Small Intestine: Report of 24 Cases. Surg., Gynec. and Obst., October, 1933, **57,** 501–507.
(29) RHINEHART: Roentgenographic Technic, 1930 Edition, p. 342.
(30) SOPER, H. W.: Roentgen Diagnosis of Lesions in the Small Intestine. RADIOLOGY, February, 1933, **20,** 76–78.

DISCUSSION

DR. PHILIP J. HODES (Philadelphia): Dr. Rowe has presented a very difficult subject in an admirable fashion. There is little to say in addition except possibly to point out several features that might be of interest in the technic used in diagnosing small intestinal lesions distal to the ampulla, and to stress certain pitfalls in such diagnoses.

This patient (first slide) was examined on March 27. The proximal jejunum and distal duodenum are situated in the midportion of the abdomen.

Examination was made of the same patient fourteen days later (second slide). The same small intestinal loops appear in another portion of the abdomen.

These slides are presented to illustrate a possibility of wrong diagnosis. Radiologists are frequently asked to point out the site of an intestinal obstruction from the plain survey abdominal film by the mere identification of distended loops. Mistakes in such opinions may occur often as the location of intestinal loops varies in different individuals and even in the same individual from day to day.

The importance of the plain water barium meal in the examination of the small intestine has been stressed repeatedly. Using such a meal, a certain type of small intestinal pattern will be obtained. The barium leaves the stomach in a continuous stream; fluoroscopically, its progress is barely perceptible. Peristaltic rushes are infrequent and bolus formations are not the rule except in the lower ileum.

Under normal conditions the stomach empties the water barium meal in about two hours, although sometimes it may take three hours. The water barium reaches the cecum in approximately from one and a half to three hours, and the entire small intestine is emptied in from four to six hours.

As other speakers have indicated, extrinsic factors such as fear, pain and, in some cases, sensitivity to barium influence the small intestinal pattern.

The importance of fluoroscopy has not been stressed sufficiently in the diagnosis of small intestinal lesions. Roentgenograms taken at hour intervals will not suffice. Fluoroscopic examinations should be made as often as fifteen or, at the most, thirty-minute intervals in such patients. This technic will enable radiologists to pick up transient changes in motility and pattern which may hold the key to a difficult diagnosis. While we recognize this procedure may be tedious, we feel that the results obtained will justify the time consumed.

THE MOBILITY OF THE ANTRUM, PYLORUS, DUODENUM, AND GALL BLADDER IN HEALTH AND DISEASE: THE INFLUENCE OF MOBILITY IN THE FUNCTIONING OF THESE ORGANS IN THE BILIARY TRACT[1]

By NATHAN B. NEWCOMER, M.D., *and* ELIZABETH H. NEWCOMER, M.D., *Denver*

IN order to discuss this subject intelligently, it is necessary to give a detailed description of the method of suspension of the organs of the upper right quadrant which permits their mobility, and also, why mobility is necessary for the proper functioning of these organs. The peculiar method of their suspension also explains why these organs can assume such varied locations in the abdomen.

It is a matter of everyday observation by roentgenologists and surgeons that the location of the stomach, pylorus, cap, and gall bladder varies with different types of habitus. In the sthenic types, the organs lie high and the gall bladder some distance from the midline; in the asthenic types, the organs lie low and the gall bladder nearer the midline. The gall bladder is attached to the liver and partakes of its mobility.

Gray's "Anatomy" states:

"The position of the pylorus in the erect living body of the male varies from 14.5 cm. above to 8 cm. below and in the female from 15 cm. above to 2.5 cm. below the interiliac line. The range of position in regard to the sagittal axis of the erect body varies in males from 8.8 cm. to the right to 2 cm. to the left of the axis. In 84 per cent it is to the right of the axis. In females the position ranges from 6 cm. to the right to 2.6 cm. to the left of the sagittal axis. In 89.5 per cent it is to the right. The most common position in both males and females is from 2.5 cm. to 5 cm. to the right."

The first portion of the duodenum or cap is covered by peritoneum and, consequently, is freely movable. It begins at the pylorus and ends at the neck of the gall bladder. Gray's "Anatomy" states:

"It is in such close relation to the gall bladder that it is usually found stained by bile after death, especially on its anterior surface."

[1] Presented before the Radiological Society of North America at the Twenty-second Annual Meeting, Nov. 30–Dec. 4, 1936, at Cincinnati.

The stomach is covered with peritoneum anteriorly and posteriorly, thus allowing complete mobility. It is continuous with the esophagus at its upper extremity, and has a fairly rigid attachment at this point. From here, the stomach is suspended to the diaphragm and liver by the lesser omentum. Gray's "Anatomy" describes this attachment as follows:

"The lesser omentum (or gastrohepatic omentum) is the duplicature which extends to the liver from the lesser curvature of the stomach and the commencement of the duodenum. It is extremely thin, and is continuous with two layers of peritoneum which cover, respectively, the antero-superior and postero-inferior surfaces of the stomach and first part of the duodenum. When these two layers reach the lesser curvature of the stomach and the upper border of the duodenum, they join together and ascend as a double fold to the porta of the liver; to the left of the porta the fold is attached to the bottom of the fossa for the ductus venosus, along which it is carried to the diaphragm, where the two layers separate to embrace the end of the esophagus. At the right border of the omentum the two layers are continuous, and form a free margin which constitutes the anterior boundary of the epiploic foramen. The portion of the lesser omentum extending between the liver and stomach is termed the hepatogastric ligament, while that between the liver and duodenum is the hepatoduodenal ligament."

Livingston states:

"The manner in which stomach becomes connected with liver is as follows: the bulk of the liver (mesodermal portion) develops within the anterior mesentery; hence this large digestive gland becomes interposed between stomach and anterior abdominal wall so that the stomach no longer appears connected with parietes but with liver. The connecting double peritoneal fold is appropriately termed the lesser omentum (ligamentum hepatogastricum). . . . . The gastrohepatic or lesser omentum extends from the entire lesser curvature of the stomach to the under surface of the liver (transverse fissure or porta hepatis)."

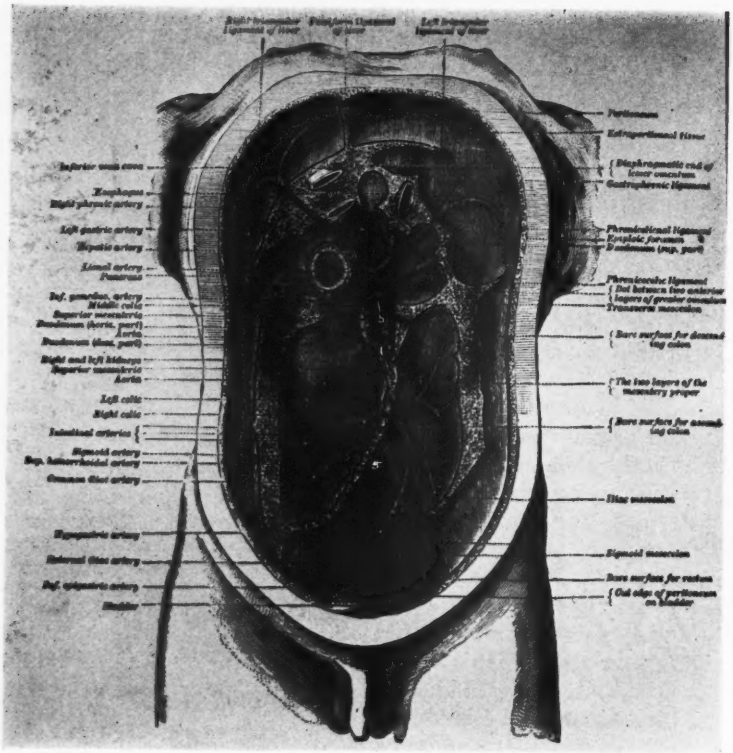


Fig. 1. See caption under Figures 2 and 3.

This suspension of the stomach to the liver allows it to move with the liver.

The liver also has a peculiar method of suspension, allowing considerable range of mobility. Cunningham's "Anatomy" describes the attachment of the liver as follows:

"Posterior Area of the Parietal Surface: (1) The uncovered area of the right lobe is a considerable portion of the back of the right lobe—varying from one and one-half to two and one-half inches in width, and from three to four inches in transverse measurement—which corresponds to the interval between the two layers of the coronary ligament, and is devoid of peritoneum. Over this uncovered portion, which looks more inward than backward, the liver and diaphragm are in perfect contact, and are united by areolar tissue."

Piersol states the following in regard to the mobility of the liver:

"Coincidently with the descent of the viscus, it undergoes a rotation or tilting forward so that its diaphragmatic surface is in contact with the abdominal wall."

This peculiar attachment of the liver allows a combination of upward and downward, and inward and outward displacement, which is accompanied by rotation. The actual position of the liver and its attached gall bladder varies markedly with the habitus; from the sthenic to the asthenic type, changes in body weight, presence of ascites, pregnancy, uterine, or ovarian tumors, etc. It undergoes a similar change in position in passing from an empty stomach to a full one.

We thus see that the position and mobility of the stomach, pylorus, and duodenal cap depend on the mobility of the liver and, likewise, the position of the gall bladder

depends on the mobility of the liver because of its attachment to it.

Complete mobility of these organs is necessary for their proper functioning. We wish to quote from Gray's "Anatomy":

"The Stomach (the Pyloric Orifice).—Its position varies with the movements of the stomach. When the stomach is empty the pylorus is situated just to the right of the median line of the body on a level with the upper border of the first lumbar vertebra. As the stomach becomes distended the pylorus moves to the right, and in a fully distended stomach may be situated two or three inches to the right of the median line.

"Alterations in Position.—When the stomach is distended, its surfaces, which are flattened when the organ is empty, become convex. The greater curvature is elevated and carried forward, so that the anterior surface is turned more or less upward and the posterior surface downward, and the stomach brought well against the anterior wall of the abdomen.

"When the stomach becomes distended the change in position of the pylorus is very considerable; it is shifted to the right, some two or three inches from the median line, and lies under cover of the liver, near the neck of the gall bladder.

"The Small Intestine (the Duodenum).—In the adult, the course of the duodenum is as follows: commencing at the pylorus, the direction of the first portion depends upon the amount of distention of the stomach and, therefore, upon the position of the pylorus. When the stomach is empty and the pylorus situated at the right of the upper border of the first lumbar vertebra, it is nearly horizontal and transverse, but when the stomach is distended, in consequence of the alteration of the position of the pylorus to the right, the proximal end of the duodenum also becomes altered in position, while the distal end remains fixed and the direction of this portion of the bowel is now anteroposterior. Whether directed transversely or anteroposteriorly, it reaches the under surface of the liver, where it takes a sharp curve and descends along the right side of the vertebral column, for a variable distance, generally to the body of the fourth lumbar vertebra."

Cunningham's "Anatomy," describing the motility and mobility of the stomach, states as follows:

"The Stomach.—With distention there comes a general enlargement of the various diameters, and elongation of the whole organ, with a consequent passage of its pyloric portion to the right beneath the liver, the development of the antrum pylori, and an inclination of its axis from behind downwards and forwards, without any rotation.

"In the gradual passage of the stomach from the empty to the distended condition we may recognize three stages.

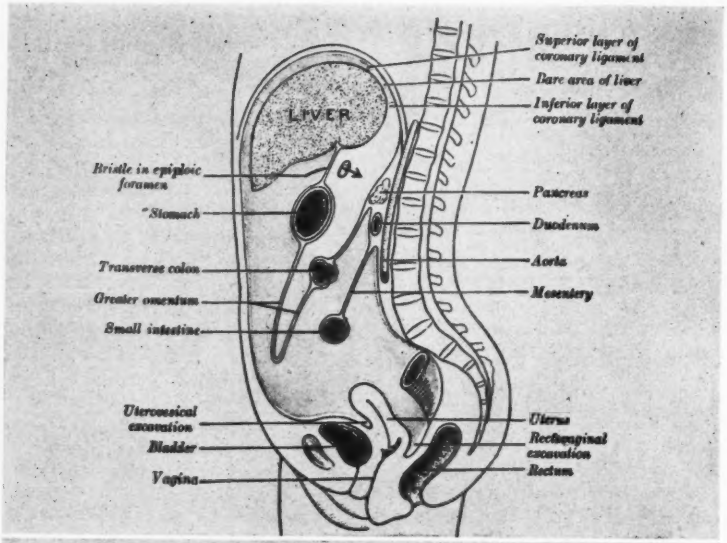
"First stage: This commences with an enlargement of the fundus, and is followed by an expansion of the whole cardiac portion, which passes upwards and also to the left towards the diaphragm, displacing the coils of the transverse colon, which lies here when the stomach is empty. The pyloric portion for three or four inches still remains contracted and cylindrical. In this condition the stomach is frequently found after death.

"Second stage: As distention goes on the lesser curvature opens out, the pyloric portion (with the exception of its last inch) expands, but its junction with the cardiac portion usually remains distinct, until distention is almost complete.

"Third stage: A further general expansion of the whole stomach takes place; the diameters of both cardiac and pyloric portions, as well as the length of the organ, are increased, and the great curvature presses forward against the anterior abdominal wall in front, where the restraining influence of the ribs is absent. The pyloric end for about one inch (2.5 cm.) from the pylorus remains narrow (constituting the pyloric canal of Jonnesco), but to the left of this it bulges forward, forming the antrum pylori, which is most distinct at the great curvature. By the increase of the organ in length the antrum is carried a considerable distance to the right beneath the liver—even farther than the pylorus itself—so that the terminal part of the stomach is bent backwards and to the left, in order to reach the pylorus, which latter very rarely passes more than one and a half or two inches to the right of its normal position, namely, in the empty condition, within half an inch (12 mm.) of the middle line.

"The narrow or pyloric end, when the stomach is empty, is contracted and cylindrical, and runs transversely to the right, lying as a rule beneath the left lobe of the liver. . . . . During distention it is carried to the right beneath the quadrate lobe, and its terminal part is there directed backwards in order to reach the duodenum. Even in this condition its last inch remains comparatively undistended.

"The Duodenum.—When the stomach is distended, the first inch of the duodenum—which is movable on account of its peritoneal covering—is carried to the right with the pylorus, and thus brought into line with the second or



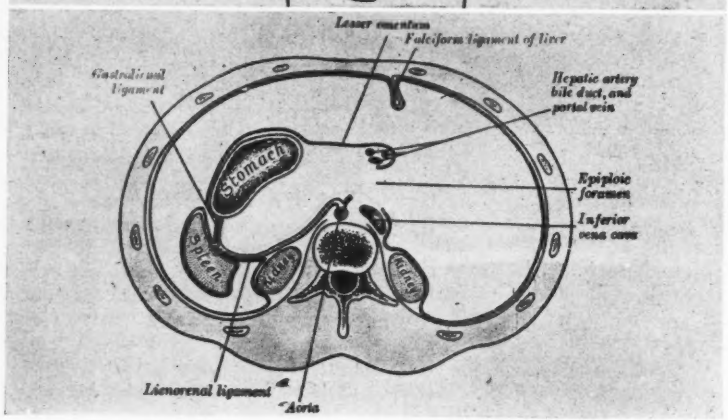


Fig. 2 (*above*). Fig. 3 (*below*).

Figs. 1, 2, and 3. (Gray's "Anatomy," 22nd Edition.) These illustrate the attachment of the liver to the posterior abdominal wall, and the attachment of the stomach to the liver, which permits the mobility of these organs.

terminal half, which is always directed backwards. Hence the whole of the first portion of the duodenum is directed backwards when the stomach is full."

In a previous article we called attention to the effect of mobility of the gall bladder on the cystic duct, and the action of the cystic duct as follows:

"The cystic duct is a hollow fibro-muscular tube lined by a redundant mucous membrane connecting the common duct with a movable organ (the gall bladder attached to the liver). Like any mechanical attachment between movable parts, it must be able to handle the play between them. After a full meal, the gall bladder is pushed upward and outward from one to two inches by the full stomach, and undergoes a certain amount of rotation because of its attachment to the liver. This straightens the cystic duct and allows it to readily empty into the common duct, as stated by Jacobson and Geydesen. We, ourselves, have repeatedly demonstrated in rabbits and guinea pigs that moderate traction on the gall bladder will straighten the cystic duct, and readily empty the gall bladder. When the stomach is empty, the muscular portion of the duct assumes some modification of the S-shape by its own con-

tractability, and the redundant mucous membrane, if rotated, would assume a spiral shape, producing the valves of Heister."

We have included this detailed description of the attachment of the organs in the upper right quadrant to show how they adjust themselves to different types of habitus, changes in body weight, pregnancy, and various pathological conditions, and especially how they adjust themselves to a full meal and to active peristalsis of the stomach.

In two previous articles we demonstrated by films and tracings from films, how the normal visualized gall bladder moved upward and outward from one and one-half to two inches after the taking of a solid meal, and also, how the intimate contact of the antrum and cap with the normal gall bladder, when the stomach was visualized, could not help but exert sufficient pressure to empty the gall bladder in the time and manner in which it is known to empty.

McMaster and Elman have shown in their work on dogs, with a tube inserted in the gall bladder, that immediately, or as they state, practically *at once* upon giving food, the bile would rise high in the tube. Boyden describes the "initial response" as follows:

"Perhaps the most striking feature of the reaction of the gall bladder to food is the very short latent period of contraction: 14 out of 17 patients showed a marked diminution of volume within the first two minutes after food entered the mouth."

We have shown how the pressure of the filled stomach and cap, in intimate contact with the gall bladdder embedded in the liver, exert sufficient pressure to displace it upward and outward from one and one-half to two inches. This is the most logical explanation of the effect on the gall bladder immediately on taking food. Repeated waves of peristalsis cause additional pressure on the gall bladder. We believe these are the main factors in emptying the gall bladder.

We have also demonstrated that if this intimate contact of the antrum and cap with the gall bladder is disturbed by overweight or under-weight, pregnancy, etc., the gall bladder will not empty but will give rise to symptoms. A careful check of the patient's history will show that a large percentage of gall bladder symptoms date back to changes in body weight. The old statement of "fair, fat, and forty" is only partly true. Of course, fat is apt to accumulate at forty. However, the gall bladder is as prone to disease in the asthenic as in the sthenic types.

Even when the gall bladder is visualized and normal in density at six hours, it is almost certain, if its mobility is limited, that is, if it does not move upward and outward from one and a half to two inches after a full meal, that it will not empty in 24 hours.

Occasionally, after giving the gall bladder dye test, which we routinely do by the intravenous method, the six-hour film will show no sign of a gall-bladder shadow. After a solid meal is given, some of these gall bladders will be visualized and are normal in shape, size, and density, and will empty in 24 hours. For several years we were at a loss to explain this peculiar condition. Why should some gall bladders visualize after a full meal but not before, be normal in density, and empty in a normal manner? Were they diseased gall bladders, and should they be removed? Since we have been paying more attention to the mobility of the organs in the upper right quadrant, we have reached the conclusion that in this type of gall bladder there is a kinking of the cystic duct which is straightened by the full meal pressing the gall bladder upward and outward, as we have described above. Many roentgenologists do not carry the test further if the gall bladder does not fill, logically enough perhaps, thinking it is foolish to give a meal to empty a gall bladder that does not fill. Furthermore, a solid meal will push the gall bladder farther upward and outward than a liquid meal. We believe that a solid meal should always be given, whether the gall bladder is visualized or not. A fat meal is absolutely unnecessary to produce empty-

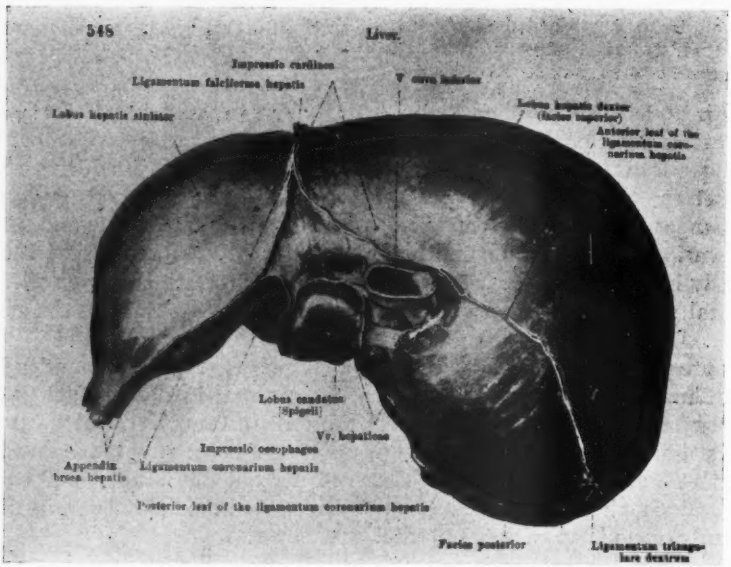


Fig. 4. (Spalteholz, Werner: "Hand-atlas of Human Anatomy," 1923.) Shows the limited amount of retroperitoneal attachment of the liver. This limited attachment gives it free mobility.

ing of a normal gall bladder, and does not even simulate normal conditions.[2]

Another problem of mobility enters into the above type of cases. Why should the gall-bladder shadow be normal in density immediately after eating? (The patient is instructed to eat a normal solid meal at some nearby restaurant, and return at once.) In our experience, it takes at least three hours for the gall bladder to concentrate the dye to a normal density.

All physiologists agree that bile is secreted more or less continuously, but more profusely during gastric digestion. What part does the pressure of the filled stomach on the liver, as described above, have on the secretion of bile, or on expressing it from the small and large biliary ducts and even from the liver cells? Apparently, in the above type of cases, the bile had been secreted and concentrated, but did not enter the gall bladder until the cystic duct was straightened by the pressure of the filled stomach against the liver. We have shown that the liver with its attached gall bladder, moves upward and outward, and rotates because of the pressure of a filled stomach on the liver. Interference with the mobility of the liver due to pathological conditions in the upper right quadrant, should have an unfavorable action on the function of the liver, if the above deductions are correct.

Many gall bladders giving symptoms, if not of too long a duration, can be restored to normal if the weight is restored to normal.

Experiments on pregnant animals have shown that the gall bladder will not function in many of them, but will function normally after delivery. The same is true of many pregnant women, if stones have not formed because of the failure of the gall bladder to empty. We believe this improvement is caused by the restoration of normal relations and free mobility of these organs.

Mobility of the Pylorus and Cap.—According to Gray's "Anatomy," as quoted

[2] In RADIOLOGY, Vol. 25, page 558, we presented films of the same individual in which barium and buttermilk emptied the gall bladder in 29 minutes, and barium and eggs and cream required one hour and fifteen minutes to empty it.

above, when the stomach is empty, the pylorus is just to the right of the median line, and when fully distended, it may be two or three inches to the right of the median line. One should always check the mobility of the pylorus during a fluoroscopic examination and, if immovable, a serious effort should be made to determine the cause. If the pylorus is always found close to the mid-line in a series of films of the filled stomach, there is usually some pathology in this region to account for it, such as adhesions, cholecystitis, ulcers, etc.

In films and under the fluoroscope, under normal conditions of mobility, the descending portion of the duodenum drops vertically. We often see the descending portion pulled to the right. The probable explanation of this is, that in inflammatory conditions in this area, the stomach will not tolerate food, and consequently, the cap is immobilized by adhesions in the empty position close to the mid-line. This gives the effect of the descending portion being pulled to the right, when, in reality, the cap is held to the left close to the mid-line.

THE PART PLAYED BY MOBILITY IN PERISTALSIS OF THE STOMACH AND DUODENAL CAP

Alvarez states:

"Another characteristic of smooth muscle in hollow organs is its responsiveness to tension. Most of the motor activities of the stomach and bowel are brought about and regulated largely by the internal pressure due to the presence of food or gas. Cannon has shown that the rhythmic segmentation in the small intestine is due simply to the fact that those muscle fibers which are stretched tend to contract. Their contraction increases the pressure in neighboring segments, and so the process goes on. Cannon has shown also that the waves in the stomach tend to appear at those places where the internal pressure balances the local tone of the muscle.

"In the human stomach, waves appear about once in twenty seconds. Carefully taken serial plates reveal the fact that these waves begin near the cardia. They travel as shallow ripples until either proper pressure conditions or the presence of the peculiar antral muscle causes them to break into deep waves. Ordinarily we see only one or two waves at a time, but in the presence of a diseased gall bladder or an ulcerated duodenum the stomach often gets so irritable that four or five deep waves course over it at one time. Reverse waves are seen only under pathological conditions. During the first stages of starvation there appear the powerful, so-called hunger contractions."

Alvarez also states:

"The gastric waves do not cross the pylorus, so far as we can see."

Cannon originally advocated the theory that the opening and closing of the pyloric sphincter was under acid control. However, this theory will not account for the emptying of the stomach in the absence of hydrochloric acid, as in achylia gastrica, and has been discarded.

Cole describes the motor phenomena of the stomach, pylorus, and cap as follows:

"The gastric motor phenomenon is complex rather than simple and is composed of the progression of the peristaltic waves pyloruswards and a systole and diastole, and it may be considered in the form of a cycle similar to the cardiac cycle. The stomach assumes a series of shapes and eventually assumes the same shape that it was approximately fifteen or twenty seconds earlier. This is referred to as a gastric cycle. . . . . During the systole of each gastric cycle, when the pressure against the proximal surface of the pyloric valve is greatest, the pyloric sphincter is partly opened, either actively or passively, and the chyme is propelled into the reservoir cap; and during the stage of diastole when the gastric pressure is diminished the sphincter may be closed to prevent the chyme from falling back into the stomach.

"The duodenum is replenished from the reservoir cap where the finishing touch of proteid digestion is applied to a small amount of chyme. The cap is evacuated by a broad peristaltic wave, independent of the gastric peristalsis, which forces the chyme through the duodenum in 'finger-like masses.' "

Wheelon and Thomas state:

"Such observations definitely establish the antrum as an organ of special motility, the purpose of which is to mix the ingesta and bring it into contact with the gastric juices, and to propel the semidigested chyme onward into the duodenum. . . . . Some time before the antrum enters upon its held contraction the sphincter begins to rapidly contract and reaches its maximum, usually at a time of marked relaxa-

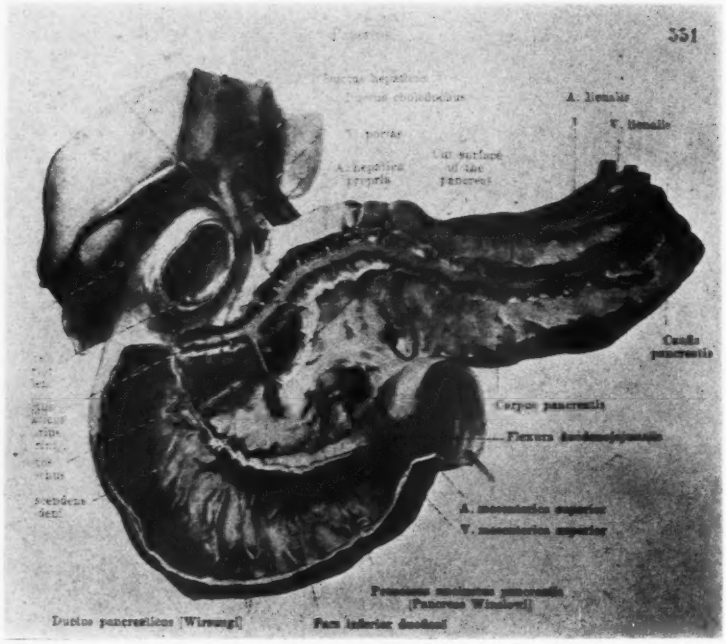


Fig. 5. (Spalteholz, Werner: "Hand-atlas of Human Anatomy," 1923.) Shows the relation of the antrum to the gall bladder and cystic duct. Pressure of a full meal pushes the gall bladder upward and outward from 1.5 to 2 inches, and straightens the cystic duct, beside exerting direct pressure upon the contents of the gall bladder.

tion of the antrum. The sphincter then relaxes and remains quiet until a second positive phase is well initiated in the antrum. Hence, the sphincter is open during the greater part of the antral contraction and actively closed while the antrum is relaxing."

They further state:

"Our results, along with those of Cole and Luckhardt, Phillips, and Carlson, demonstrate that the activities of the pyloric sphincter, at least in great part, are dependent upon the activities of the antrum. That is, the impulse to contract in the antrum during digestive processes is propagated into the sphincter, thereby causing it to contract at a time of relaxation of the antrum and to relax because of the arrival of a wave of relaxation over the antrum. Such conditions indirectly lead to the conclusion that acid if it acts to regulate the 'pylorus' must also act in a similar way upon the antrum and stomach as a whole, for, as shown above, motility of the antrum determined motility of the sphincter."

Starling states:

"The wave as it travels along exhibits every two to three seconds the rhythmic fluctuations in intensity common to most plain muscle contractions, and when a wave at a phase of increase reaches a point about one inch from the pyloric canal it is so marked that part of the pyloric antrum becomes almost completely separated from the rest of the stomach. The part thus cut off then diminishes in size in every direction, part of its contents being forced through the pyloric canal, while the remainder escapes back as an axial reflux stream into the stomach. The waves recur at regular intervals of fifteen to twenty seconds, and three or four are present simultaneously. They continue without cessation until the stomach is empty—from one to four hours after the meal, according to its bulk and composition.

"It has indeed been thought that the acidity of the gastric contents exercises a direct inhibitory action on the pyloric sphincter, but recent work has shown that this is not the case. It appears more likely that the onward passage of gastric contents depends on two factors, first, the relaxation of the pyloric sphincter, and second, the relative pressures in the pyloric antrum and the first part of the duodenum

(duodenal cap). When a wave in an intense phase reaches the pyloric antrum, the sphincter relaxes before it, and as the pressure in the antrum is high, its contents escape into the duodenum. The contraction wave then involves the sphincter and closes it."

In 1911, Cole concluded that pathology in the upper right quadrant had its effect on peristalsis. He states:

"They do, however, represent various types of gastric peristalsis in stomachs that show no radiographic evidence of a pathological lesion in or around the stomach or duodenum, and are, therefore, called 'unobstructed,' in contradistinction to those in which the peristaltic contraction or wave is obstructed by early carcinoma, ulcers, or adhesions from gall-bladder infection."

Luckhardt, Phillips, and Carlson state:

"Fluoroscopic examination of the stomach of man while recording simultaneously graphically the motor activity of the stomach shows that the pylorus opens for the ejection of chyme with arrival at the pylorus of powerful advancing rings of constriction aided possibly by a general increase in tone of the stomach musculature as a whole."

Cole, in discussing adhesions, states:

"The manner in which the cap lies between the gall bladder on the right and the common duct on the left, and the fact that slight adhesions prevent its normal dilatation, must always be borne in mind.

"Adhesions are caused by gastric or duodenal ulcer, or gall-bladder infection, with or without calculi, in a large percentage of cases, and therefore they generally indicate that one of these conditions is present or has existed. From the location and character of the adhesions one can sometimes tell which of these conditions has caused them."

Lack of expansion of the antrum is often seen in gall-bladder disease. George and Leonard state:

"There is, however, one type of spasm that in our experience is so commonly associated with gall-bladder disease that we have come to consider it when present as fairly reliable evidence. This type of spasm affects the antrum of the stomach. Usually the distal third of the stomach becomes uniformly contracted, producing a tubular outline an inch or so in diameter, the proximal two-thirds maintaining its normal diameter."

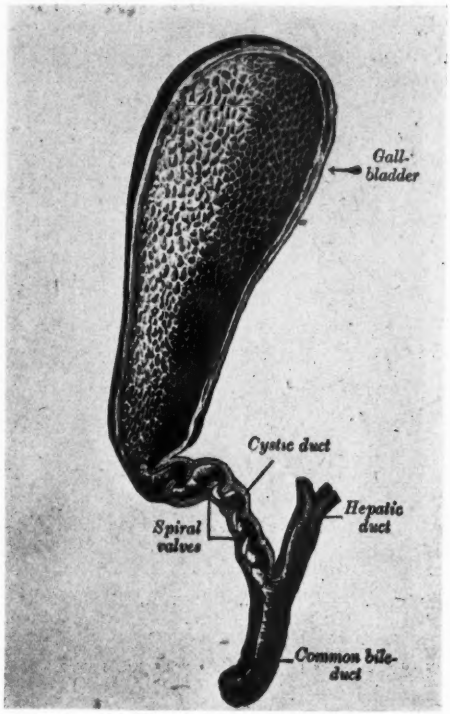


Fig. 6. (Spalteholz, Werner: "Hand-atlas of Human Anatomy," 1923.) Shows the convolutions of the cystic duct. When the gall bladder moves upward and outward from 1.5 to 2 inches after a full meal, by straightening of the convolutions, the duct permits this range of motion.

Carman stated:

"Spasms of the gastric musculature occur frequently in association with cholelithiasis, cholecystitis and pericholecystitis—so frequently that the gall bladder should be suspected first of all whenever spastic eccentricities of the stomach are noted. The spasmodic manifestations in the stomach may be slight or extensive, ranging from a transient incisura or a temporary hour-glass to a regional spasm with considerable deformity. An especially striking type of the latter is that in which the entire pars pylorica is contracted to a narrow, stiffened, sometimes palpable, tube."

From the above review of gastric peristalsis, it is evident that distention will produce contraction. Contraction of a segment produces distention in the succeeding segment, followed by its automatic contraction. When this wave reaches the pylorus, it distends the muscles entering

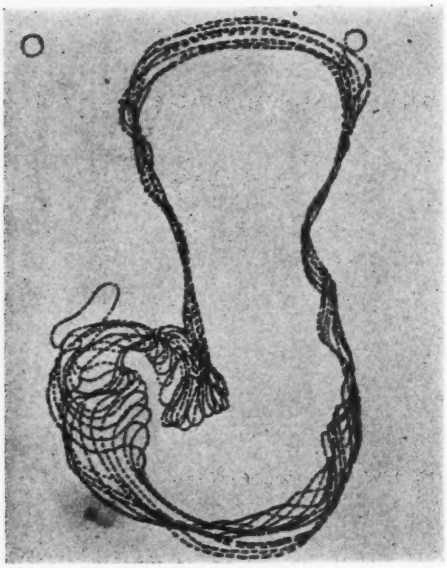


Fig. 7. (Kaestle, C., Rieder, H., and Rosenthal, Phil J.: Arch. Roentgen Ray, 1911, **15**, 3.) Shows the range of motion of the stomach and particularly the antrum, during peristalsis. Any limitation of this mobility interferes with normal peristalsis and emptying of the stomach.

into it, allowing some food to pass, and after distention, it automatically contracts the same as the other segments of the stomach. Peristaltic waves are not transmitted from the stomach to the duodenum. Consequently, new peristaltic waves arise in the cap by the same method. When sufficient food has passed through the pylorus into the cap to produce distention or dilatation, peristaltic waves will be originated there. Stretching, due to distention of the smooth muscle, is a fundamental part of peristalsis, and requires considerable freedom of motion along the axis of transmission. In the antrum the waves are especially deep, and all roentgenologists have observed the normal antrum bulge and swing to the right during active peristalsis. Freedom of motion and room for expansion are necessary for active peristalsis. Too little attention has been paid to the inhibition of gastric peristalsis due to immobility in this region.

Gastric Retention.—David M. Beckman, in summarizing gastric retention in cases operated on at the Mayo Clinic in 1922, says that, in 582 patients operated on for duodenal ulcer, gastric retention was present in 93 cases (16 per cent), and in 97 cases operated on for gastric ulcer, gastric retention was present in 22 (22.6 per cent). In the 115 cases of gastric retention, actual narrowing or obstruction of the pyloric ring was demonstrated at operation in only 22.7 per cent of the gastric ulcers and 46 per cent of the duodenal ulcers.

Eusterman and Balfour report in 778 surgically verified cases of duodenal ulcer at the Mayo Clinic, gastric retention or pyloric obstruction was recorded in 20 per cent. In 324 cases of chronic benign gastric ulcer, retention was noted in 29 per cent. In 35 per cent of the cases in which the site was definitely recorded by the surgeon, the gastric ulcer was remote from the pylorus.

In our experience, gastric retention of from one-third to one-fourth is a very common complication of gall-bladder disease. In a great many cases, even though there is no gastric retention of food, there is a scattering of the meal through the jejunum and proximal loops of the ileum, showing a definite delay in leaving the stomach. Gall-bladder disease definitely inhibits peristalsis as is seen by the long, narrow antrum and lack of dilatation of the antrum, so long noted by roentgenologists in many cases of advanced gall-bladder disease.

We have shown in previous articles that, if the gall bladder does not move upward and outward from one and one-half to two inches after a full meal of solid food, the gall bladder will not empty in twenty-four hours; and *vice versa*, if it does have the above mobility, it will empty normally.

We have seen several cases of gastric retention following cholecystectomy that were not present before operation. We have also seen other cases of gastric retention, with adhesions to the antrum, which did not cause any narrowing of the antrum. We believe many adhesions cause delay in emptying the stomach, by interfering with

the mobility and dilatation of the antrum, thus inhibiting peristalsis.

Normal peristalsis in the antral portion of the stomach consists of vigorous waves, of contraction and dilatation, both of which are essential to peristalsis. The wave is started by over-stretching. The contraction of a segment causes the succeeding portion to expand, which automatically contracts because it has been stretched. This is accompanied by a marked displacement of the antrum and pylorus to the right, which requires considerable range of motion or mobility. It is the inherent nature of an involuntary muscle that stretching and expansion are necessary before it can contract. Thus, anything interfering with its expansion and mobility will inhibit peristalsis. Vigorous contractions of the antrum are necessary to overcome the pyloric sphincter, and any inhibition of gastric peristalsis will delay emptying of the stomach, even though there is no organic narrowing of the pyloric sphincter.

Distention of the cap is, likewise, necessary to initiate peristalsis in the duodenum, and free mobility is necessary for its proper function.

Alvarez says:

"The duodenal cap tends to remain filled during the early stages of digestion. This may be due to the low rhythmicity of the muscle in that region, to its thinness and weakness (in man), to its firm attachment to the mucous membrane, and perhaps to the peculiar festoon-like arrangement of the fibers."

May we suggest a probable explanation as to why the cap tends to remain full? It requires a certain amount of distention to initiate a peristaltic wave. It requires several gastric peristaltic waves to distend the cap sufficiently, so that it will initiate a peristaltic wave. When this occurs, the cap is evacuated at once. Of necessity the cap would be filled, or partly filled, the major portion of the time, as it empties quickly and fills slowly.

CONCLUSION

We have shown how the suspension of the stomach and duodenal cap to the liver, and the attachment of the liver by its posterior and inner border to the diaphragm, permit the marked mobility of these organs which is so necessary for their proper functioning. We have shown how limitation of this mobility interferes with the function of the liver, gall bladder, biliary duct system, stomach, and duodenum.

BIBLIOGRAPHY

1. ALVAREZ, WALTER C.: The Mechanics of the Digestive Tract. 1922.
2. IDEM: The Mechanics of the Digestive Tract. Second Edition, 1929.
3. ALVAREZ, WALTER C., and ZIMMERMANN, A.: Movements of the Stomach. Am. Jour. Physiol., 1928, **84,** 261.
4. BERKMAN, DAVID M.: Gastric Retention: Its Treatment and the Surgical Mortality. Collected Papers of Mayo Clinic and Mayo Foundation, 1923, **15.**
5. BOYDEN, E. A.: An Analysis of the Reaction of the Human Gall Bladder to Food. Anat. Rec., 1928, **40,** 147.
6. IDEM: Study of the Behavior of the Human Gall Bladder in Response to the Ingestion of Food: Together with Some Observations on the Mechanism of the Expulsion of Bile in Experimental Animals. Anat. Rec., 1926, **33,** 201.
7. IDEM: Behavior of the Human Gall Bladder during Fasting and in Response to Food. Proc. Soc. Exper. Biol. and Med., 1926, **24,** 157.
8. CANNON, WALTER B.: Some Reflections on the Digestive Process. Caldwell Lecture, 1934. Am. Jour. Roentgenol. and Rad. Ther., 1934, **32,** 575.
9. IDEM: The Acid Control of the Pylorus. Am. Jour. Physiol., 1907, **19–20,** 283.
10. IDEM: The Influence of Emotional States on the Functions of the Alimentary Canal. Am. Jour. Med. Sci., 1909, **137,** 480.
11. IDEM: The Movements of the Stomach Studied by Means of the Roentgen Rays. Am. Jour. Physiol., 1898–1899, **1–2,** 359.
12. IDEM: The Nature of Gastric Peristalsis. Am. Jour. Physiol., 1911–1912, **29–30,** 250.
13. CAPPS, JOSEPH A.: An Experimental and Clinical Study of Pain in the Pleura, Pericardium, and Peritoneum. 1932.
14. CARMAN, R. D.: The Roentgen Diagnosis of Diseases of the Alimentary Canal. 1920.
15. COLE, LEWIS GREGORY: Motor Phenomena of the Stomach, Pylorus, and Cap Observed Roentgenographically. Am. Jour. Physiol., 1916–1917, **41–42,** 618.
16. IDEM: Serial Radiography in the Differential Diagnosis of Carcinoma of the Stomach, Gall-bladder Infection, and Gastric or Duodenal Ulcer. Arch. Roentgen Ray, October, 1912, p. 172.
17. IDEM: The Complex Motor Phenomena of Various Types of Unobstructed Gastric Peristalsis. Arch. Roentgen Ray, December, 1911, p. 242.
18. IDEM: The Diagnosis of Post-pyloric (Duodenal) Ulcer. Lancet, 1914, **1,** 1239.
19. CUNNINGHAM, DANIEL JOHN: Text-book of Anatomy, 1905.
20. ELMAN, ROBERT, and MCMASTER, PHILIP D.: Physiological Variations in Resistance to Bile Flow to the Intestine. Jour. Exper. Med., 1926, **44,** 151.
21. EUSTERMAN, GEORGE B., and BALFOUR, DONALD C.: The Stomach and Duodenum. 1935.
22. GEORGE, ARIAL W., and LEONARD, RALPH D.: The Pathological Gall Bladder. Annals of Roentgenol., 1922, **2.**

23. GIANTURCO, CESARE: Some Mechanical Factors of Gastric Physiology. Study I.—The Empty Stomach and its Various Ways of Filling. The Pressure Exerted by the Gastric Walls on the Gastric Content. The Physical Changes Occurring to the Foodstuff during Digestion. Am. Jour. Roentgenol. and Rad. Ther., 1934, **31,** 735.

24. IDEM: Some Mechanical Factors of Gastric Physiology. Study II.—The Pyloric Mechanism: The Effect of Various Foods on the Emptying of the Stomach. Am. Jour. Roentgenol. and Rad. Ther., 1934, **31,** 745.

25. GIANTURCO, CESARE, and ALVAREZ, W. C.: Roentgen-ray Motion Pictures of the Stomach. Proc. Staff Meet., Mayo Clinic, 1932, **7,** 669.

26. GRAY, HENRY: Anatomy (Edited by W. H. Lewis), 1930.

27. HEDBLOM, CARL A., and CANNON, WALTER B.: Some Conditions Affecting the Discharge of Food from the Stomach. Am. Jour. Med. Sci., 1909, **138,** 504.

28. IVY, A. C.: Contributions to the Physiology of the Stomach. Studies in Water Drinking. Am. Jour. Physiol., 1918, **45–46,** 420.

29. IVY, A. C., and SANDBLOM, PHILIP: Biliary Dyskinesia. Ann. Int. Med., 1934–1935, **8,** 115.

30. JACOBSON, CONRAD, and GYDESEN, CARL: Function of the Gall Bladder in Biliary Flow. Arch. Surg., 1922, **5,** 374.

31. JORDAN, ALFRED C.: Chronic Intestinal Stasis. 1923.

32. JUDD, E. STARR, and MANN, FRANK C.: Effect of Removal of the Gall Bladder. Surg., Gynec., and Obst., 1917, **24,** 437.

33. KAESTLE, RIEDER H., and ROSENTHAL, PHIL. J.: The Bioroentgenography of the Internal Organs. Arch. Roentgen Ray, 1911, **15,** 3.

34. KEITH, ARTHUR: A New Theory of the Causation of Enterostasis. Lancet, 1915, **2,** 371.

35. LIVINGSTON, EDWARD MEAKIN: A Clinical Study of the Abdominal Cavity and Peritoneum. 1932.

36. LUCKHARDT, A. B., PHILLIPS, H. T., and CARLSON, A. J.: Contributions to the Physiology of the Stomach: The Control of the Pylorus. Am. Jour. Physiol., 1919, **49–50,** 57.

37. MENTZER, STANLEY H.: Valves of Heister. Arch. Surg., 1926, **13,** 511.

38. MOYNIHAN, SIR BERKELEY: Addresses on Surgical Subjects. 1928.

39. IDEM: Two Lectures on Gastric and Duodenal Ulcer. 1923.

40. NEILSON, CHARLES HUGH, and LIPSITZ, SAMUEL T.: The Effect of Various Procedures on the Passage of Liquids from the Stomach. Jour. Am. Med. Assn., March 27, 1915, p. 1052.

41. NEWCOMER, NATHAN B., and NEWCOMER, ELIZABETH: Further Discussion of the Relations of the Antrum and Cap to the Gall Bladder as Factors in Emptying the Gall Bladder. RADIOLOGY, 1935, **25,** 547.

42. NEWCOMER, NATHAN B., NEWCOMER, ELIZABETH, and CONYERS, C. A.: A Proper Gastro-intestinal Examination. Colo. Med., July, 1930.

43. IDEM: Conclusions Based upon the Routine Intravenous Administration of Sodium Tetraiodophenolphthalein in Gall-bladder Diagnosis. RADIOLOGY, July, 1928, **11,** 56.

44. PFAHLER, G. E.: The Diagnosis of the Size, Form, Position, and Motility of the Stomach and Bowel by Means of the X-ray. Arch. Physiologic Therapy, July, 1905, **6.**

45. POPE, CHARLES E: Effect of Gastro-intestinal Operations on the Emptying of the Gall Bladder. Am. Jour. Med. Sci., July, 1929, **178,** 48.

46. REHFUSS, MARTIN E., BERGEIM, OLIF, and HAWK, PHILIP B.: Gastro-intestinal Studies. I.—The Question of the Residuum Found in the Empty Stomach. Jour. Am. Med. Assn., July, 1914, **11.**

47. SMITH, FRED M., and MILLER, GEORGE H.: The Reflex Influence of the Colon, Appendix, and Gall Bladder on the Stomach. Arch. Int. Med., 1930, **46,** 988.

48. SPALTEHOLZ, WERNER: Hand-atlas of Human Anatomy. 1923.

49. STARLING, ERNEST H.: Principles of Human Physiology, Fifth Edition, 1930, p. 569.

50. WEIR, JAMES F., and SNELL, ALBERT M.: Symptoms which Persist after Cholecystectomy: Their Nature and Probable Significance. Collected Papers of Mayo Clinic and Mayo Foundation, 1935, **27,** 119.

51. WHEELON, HOMER, and THOMAS, J. EARL: Observations on the Motility of the Antrum and the Relation of Rhythmic Activity of the Pyloric Sphincter to that of the Antrum. Jour. Lab. and Clin. Med., 1920–1921, **6,** 124.

52. IDEM: Rhythmicity of the Pyloric Sphincter. Am. Jour. Physiol., 1920–1921, **53–54,** 560.

53. WHITAKER, L. R., and EMERSON, W. C.: Emptying of the Gall Bladder in Pregnancy. Am. Jour. Physiol., 1928, **84,** 516.

DISCUSSION

DR. ALFRED L. L. BELL (Brooklyn, N. Y.): Dr. Newcomer has covered very thoroughly in his paper all of the mechanical aspects of mobility and motility in gall-bladder function.

I think we are all dissatisfied with the present theories with regard to biliary tract function, particularly with regard to gall-bladder emptying, and anything which will serve to clear up the doubts which we have had concerning these theories helps considerably.

Dr. Newcomer has stressed one factor which is a very valuable one. However, I feel that we cannot exclude all other possibilities with regard to gall-bladder emptying in favor of the mechanical theory.

We know, for instance, that the gall bladder has a very complete musculature and that this musculature is properly innervated. We know that under certain circumstances, at least, this musculature seems inadequate to produce proper emptying, and we know that emptying does take place, or at least intra-colicystic pressure increases, experimentally, after ingestion of a meal even when all nerves to the gall bladder have been severed.

We are all dissatisfied, I think, with the absorption theory of gall-bladder emptying, that is, that bile is absorbed only from the gall bladder and is not emptied at all by the cystic duct. Bile salts have been found in the vessels from the gall bladder but

this is not proof of the fact that all bile is evacuated by this means.

Failure of the gall bladder to function properly with changes of weight in pregnancy, while it is common, is not an absolutely constant finding, and I think that we cannot attribute all of the biliary tract difficulties which these patients have to the increase of weight in the pregnancy, and, therefore, to mechanical factors.

There are some questions which I think can be raised concerning this mechanical theory as an all-inclusive one. For example, the cases of gastro-enterostomy in which pyloric stenosis is present and in which the food does not enter—at least does not distend—the pyloric antrum. Certainly all of these cases do not show biliary tract pathology. As a matter of fact, I recently have seen one in which the dye test showed perfectly normal visualization of the gall bladder and normal emptying and yet no part of the meal in the upper part of the intestinal tract approached the gall bladder.

I feel, therefore, that while these mechanical factors which Dr. Newcomer has described are very valuable factors and probably are the missing link that we have been looking for, still we must take into consideration all other factors with regard to the emptying of the gall bladder.

DR. NATHAN B. NEWCOMER (closing): I wish to thank Dr. Bell for his discussion.

In regard to the musculature of the gall bladder, it is very ineffectual. The gall bladder will empty with or without innervation in the presence of food. The main thing that stands out in emptying of the gall bladder is its emptying when food is taken. A fat meal is unnecessary in emptying the gall bladder.

Several authorities have shown that this does not occur slowly; the major response occurs at once, within a minute or two, and I have tried to show you what happens in the mechanical relations of the gall bladder and stomach during that minute or two.

Immediately upon filling the stomach, it moves over to the right and up. It pushes the gall bladder up and to the right about two inches. There is an intimate contact between the antrum and cap, and the gall bladder, causing local external pressure on the gall bladder. If I had had time to show my slides, I would have shown that a normal gall bladder will be contacted by the antrum and cap.

This first rise in pressure, that McMaster and Elman describe, occurs immediately when the gall bladder is pressed upon by the filled stomach, including the antrum and cap. The initial response occurs in two minutes, and this is what happens during that two minutes: The stomach pushes firmly against the gall bladder and cap and subsequently peristalsis increases this pressure. I do not claim this is the only factor in emptying. In the cases of gastro-enterostomy and gastrectomy, it is very questionable whether the gall bladder will stay normal over a period of time because of the alteration in the relations of these organs following the operation.

Several authorities have shown that the greatest response to food in the stomach occurs within a minute or two after food has entered the stomach, and we have shown what mechanical changes occur in the relations of the stomach in that time.

AN ATTEMPT TO CASTRATE THE CHICK EMBRYO WITH X-RAYS

By J. M. ESSENBERG, PH.D., *Chicago*

From the Department of Anatomy, Loyola University School of Medicine

INTRODUCTION

THE theory of early germ cell segregation in animals was advanced by Nussbaum, in 1880. Swift (7) contended that the primordial germ cells in the chick embryo arise extra-embryonally during the primitive streak stage from an area known as the germinal crescent. The large entodermal cells enter the adjacent mesoderm and pass to the gonadal region. As such, they enter the germinal epithelium and become the definitive germ cells of the chick embryo. Benoit (1) attempted to destroy the germinal crescent by ultra-violet irradiation. Chick embryos from 18 to 24 hours old were irradiated for from 10 to 15 minutes and then allowed to incubate for about two days. Histologic examination revealed that there were no germ cells in the gonadal region. Essenberg and Garwacki (4) reported the finding of transitional stages in germ cells, indicating that germ cells originate from the peritoneal epithelium in the chick embryo. These conflicting data induced the present writer to undertake the study of the effect of x-rays on the germinal crescent.

MATERIALS AND METHODS

Eggs of white and brown leghorn chickens were used for this experiment. They were irradiated at 16, 18, 20, 22, and 24 hours of incubation, each group being treated with 400, 500, and 600 units of x-rays. After irradiation the eggs were permitted to incubate at the ages of two, three, and six days. The material was fixed in Bouin's fluid, embedded in paraffin, and sectioned from 6 to 8 microns in thickness. Heidenhain's iron hematoxylin was used for staining.

The x-ray machine[1] used was mechanically rectified and provided with a Landauer roentgenometer. A universal Coolidge therapy tube was used. The set-up of the machine for the entire experiment was as follows: The kilovolt meter was set at 96, which delivered 112 peak kilovolts as measured by the sphere gap; the focal distance was 10 inches; the filter was equivalent to 4 mm. aluminum; the roentgenometer was kept at 3.2 microamperes, which, by calculation, gave 0.6 r per second. The desired r, or dosage, was obtained by varying the time of exposure.

A specially designed irradiation box (4), was used for this experiment. In it was incorporated a portable incubator, a candler, and a scattered-ray absorber.

[1] "Type C" model, made by the Standard X-ray Company, Chicago.

RESULTS

Embryos Two Days Old.—In this group are included embryos from 24 to 32 hours after irradiation. Specimens treated with lethal doses of x-rays show advanced cellular decomposition. Embryos, having received non-lethal dosages, show definite signs of recovery; mitosis takes place, and cells assume a more normal appearance, although pycnotic and degenerating cells are numerous. Unmistakable primordial germ cells have not been identified in irradiated chick embryos. In this stage of development the lateral wings of the splanchnic mesoderm have not fused to form the dorsal mesentery, yet there is a definite indication of formation of germinal epithelium.

Chick Embryos Three Days Old.—Embryos irradiated with lethal doses are in advanced stages of degeneration. Embryos treated to non-lethal dosage may be divided into two classes: one that presents various degrees of malformation, and the other in which the embryos are, to all appearances, normally formed. The abnormalities may be of various kinds: from the lack of a digit to the lack of whole parts

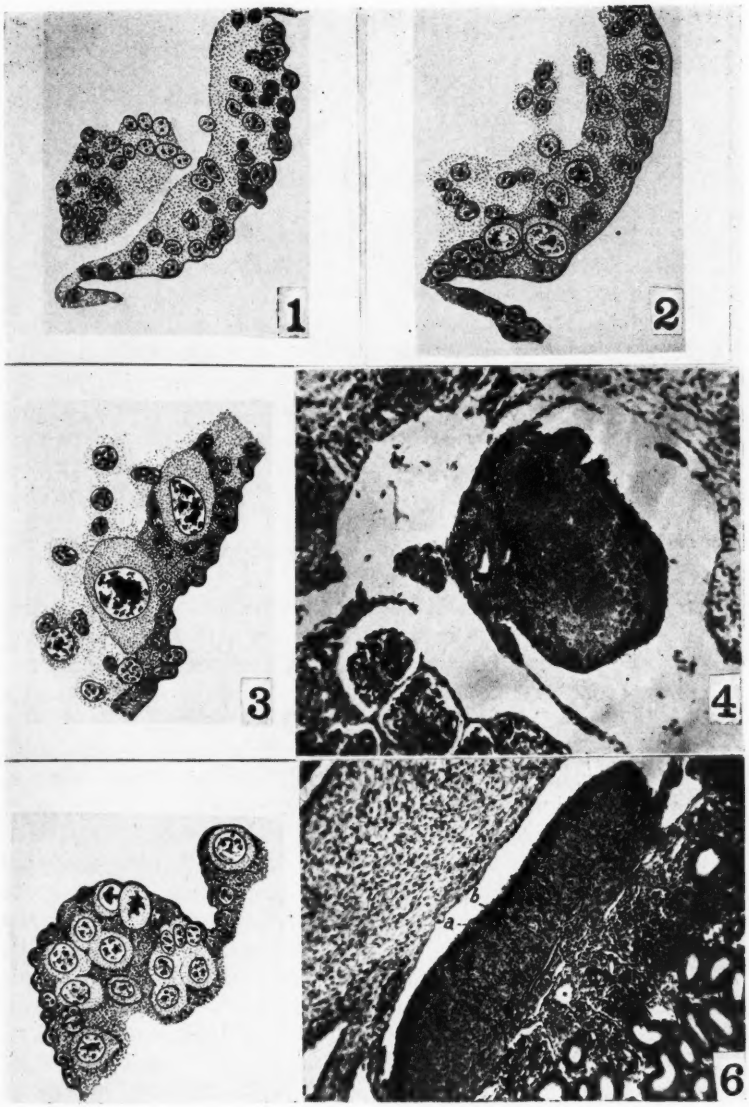


Fig. 1. Section through the germinal epithelium of a three-day-old chick embryo irradiated with 550 r at 20 hours' incubation. Note the presence of transitional germ cell stages and the absence of fully formed germ cells (× 950).

Fig. 2. Section through the germinal epithelium of a three-day-old chick embryo irradiated with 600 r at 22 hours' incubation. Note the presence of both transitional stages and fully formed germ cells (× 950).

Fig. 3. Section through the germinal epithelium of a three-day-old chick embryo irradiated with 550 r at 20 hours' incubation. Note giant germ cells (× 950).

Fig. 4. Transverse section through a testis of a six-day-old chick embryo irradiated with 600 r at 18 hours' incubation. Note germinal epithelium with fully formed germ cells and testis cords (× 120).

Fig. 5. Section through the germinal epithelium of the same testis as shown in Figure 4 (× 950). Note fully formed germ cells and transitional stages.

Fig. 6. Transverse section through the ovary of a six-day old chick embryo irradiated with 550 r at 20 hours' incubation. Note fully formed germ cells at *a* and *b* (× 120).

of the body (2). In the normally formed chick embryos, the germinal epithelium is in the process of formation. Early stages of germ cell transformation from peritoneal epithelium have been found in all specimens studied (Fig. 1). However, whether fully formed or precocious, germ cells are seldom found in three-day-old chick embryos. They may be found in a few places of the gonad, as shown in Figure 2, but there are embryos in which none are found. Occasionally, unusually large germ cells, from three to four times the normal size, are encountered (Fig. 3). These have the appearance of precocious oöcytes; they have not been observed in normal material at this early stage of development and are probably the result of the x-rays.

A Six-day-old Embryo having Received a Non-lethal Dose.—The gonads at this stage of development do not appear to differ from the non-irradiated ones except in size. On the average, the treated are slightly smaller than the non-treated gonads (Figs. 4 and 6). The germinal epithelium is thick and contains not only fully formed germ cells, but all the intermediate stages as well (Figs. 5 and 7). The medulla is composed of cords in which lumina have already formed (Fig. 4), and in which all the cell types, including fully formed germ cells, are found (Figs. 6 and 7).

A Six-day-old Embryo having Received a Lethal Dose.—This specimen was alive when the shell was opened but presented marked malformations; it was more like a lump of rarified tissue than a chick embryo. In sections, some organs, such as the neural tube, mesonephros, and the gonads, could be readily identified (Fig. 8). In the gonads, undoubtedly the testes, the germinal epithelium, and the testis cords can be recognized, but the tissues are far from being normal. In both places the cells are few and far apart (Fig. 9). However, unmistakable germ cells occur in both germinal epithelium and testis cords. Among these are fully formed germ cells as well as the transition stages (Fig. 10).

DISCUSSION

From former work (2), it is known that, with the present set-up of the x-ray machine and age of the material, 600 r is lethal to about 96 per cent of all chick embryos irradiated. Most of these die a few hours after exposure to x-rays, but some will survive for several days, and a few will hatch with or without apparent deformities. The factors involved in the production of this wide variation in results must, for the present, be explained only on general principles. The quality and quantity of the x-rays is not without variation no matter how accurately the machine is set (Packard, 6). This, however, appears to be of minor importance because the eggs were irradiated in triplets and, as a rule, the development of each was different. It is clear that hereditary and environmental factors prior to laying play the major rôle.

The principle of Bergonie and Tribondeau that the more embryonal or undifferentiated the type of cell, the greater is its radiosensitivity, is well established. The chick embryos were irradiated when the primordial germ cells were splitting off from the germ wall entoderm and undergoing their early differentiation. They were cytogenetically younger and less differentiated than those of the primitive ectoderm, entoderm, and mesoderm, and, therefore, should have been destroyed first. This indeed must have happened for none could be identified in the x-rayed chick embryos. It is thought significant that definitive germ cells occur in specimens irradiated with lethal doses.

Germ cells were present in all irradiated embryos of six days' incubation; they may be found in younger embryos but only in a few specimens and then in not very large numbers. This may be due to the fact that in the early stages the x-rayed embryos lag behind the normals in development. The germinal epithelium is well advanced in a three-day chick embryo and when intermediate cell stages are abundant. These facts lend support to the contention that the cells of the germinal crescent have

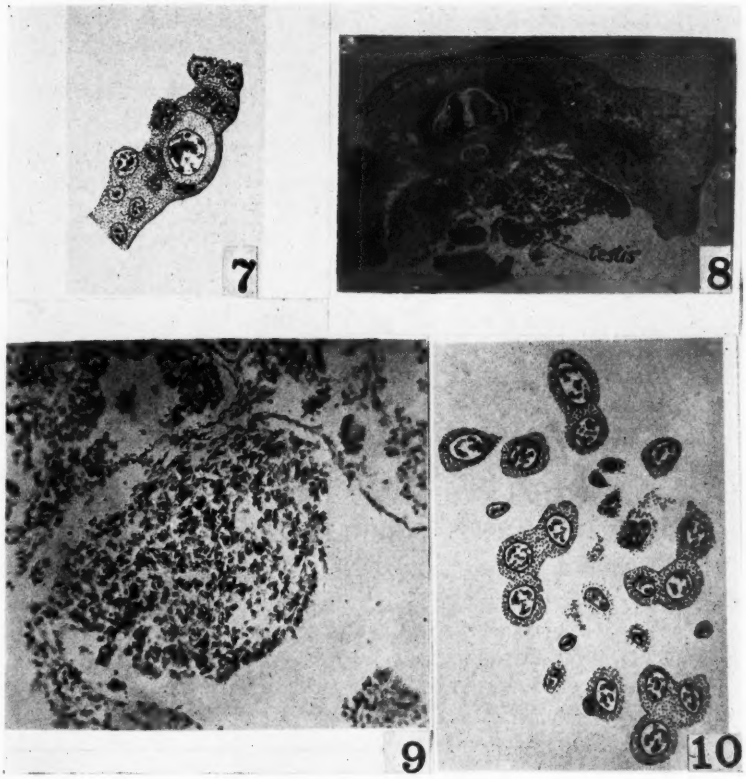


Fig. 7. Section through the germinal epithelium of the same ovary as shown in Figure 6 (× 950). Note fully formed germ cell and transitional stages.
Fig. 8. Transverse section through the gonadal region of six-day-old chick embryo having received a lethal dose of x-rays. Note testis (× 20).
Fig. 9. The same section as shown in Figure 8, with testis (× 100). Note rarified tissues.
Fig. 10. The same section as shown in Figure 8 (× 950). Note fully formed germ cells and transitional stages.

no direct relation to the formation of definitive germ cells in the chick embryo. These originate *de novo* from cells of the peritoneal epithelium. It is also believed that the above facts explain Benoit's findings. His chick embryos were too young, about 56 to 72 hours, to find fully formed germ cells, and his material was probably not sufficiently abundant to detect the difference.

SUMMARY AND CONCLUSIONS

The eggs of domestic hens were x-rayed at the primitive streak stage of development with 400, 500, and 600 r and allowed to incubate at the ages of two, three, and six days. The findings are as follows:

(1) With the set-up of the x-ray machine used in this experiment and the embryos between 16 and 24 hours of age, 600 r is lethal to about 96 per cent of all embryos irradiated.

(2) Most of the embryos irradiated with a lethal dose die shortly after irradiation; some survive for several days.

(3) Embryos irradiated with a non-lethal dose show definite signs of recovery 24 hours after treatment.

(4) In embryos three days of age, the germinal epithelium is in formation. Fully formed germ cells are seldom found; intermediate steps are numerous.

(5) With the exception of a slight reduc-

tion in size, the gonads of six-day-old embryos irradiated with a non-lethal dose appear to be normal. The germinal epithelium is developed; fully formed germ cells as well as the intermediate stages are abundant.

(6) In a six-day-old embryo, having received a lethal dose, marked cellular destruction has occurred in all organs, including the gonads, yet unmistakable germ cells are present.

(7) The migratory entodermal cells or primordial germ cells of other authors have not been identified in irradiated material.

(8) The present material supports the conception that the cells of the germinal crescent have no direct relation to the formation of the definitive germ cells in the chick embryo.

(9) The origin of definitive germ cells from the peritoneal epithelium is supported.

LITERATURE CITED

(1) BENOIT, V.: Destruction des gonocytes primaires dans le blastoderme du poulet par les rayons ultraviolets, aux premiers stades du développement embryonaire. Proc. Second Internat. Congress for Sex Research, 1930, p. 162.

(2) ESSENBERG, J. M.: The Effect of X-rays on the Developing Chick. RADIOLOGY, 1935, **25**, 739–744.

(3) Idem: A Simple Device for X-raying Eggs of the Domestic Fowl. RADIOLOGY, October, 1936, **27**, 490.

(4) ESSENBERG, J. M., and GARWACKI, JOHN H.: The Origin and Development of the Definitive Germ Cells in the Domestic Fowl. Anat. Rec., 1935, **61**, 16 (abstract).

(5) NUSSBAUM, M.: Zur Differenzierung des Geschlechts in Tierreich. Arch. f. mikr. Anat., 1880, **18**, 1–121.

(6) PACKARD, CHARLES: The Measurements of Quantitative Biological Effects of X-rays. Jour. Cancer Research, 1926, **10**, 319–339.

(7) SWIFT, C. H.: Origin and Early History of the Primordial Germ Cells of the Chick. Am. Jour. Anat., 1914, **15**, 482–516.

X-RAY DIFFRACTION STUDIES OF GLOBULAR PROTEINS

III. THE ACTION OF FORMALDEHYDE ON PROTEINS

By GEORGE L. CLARK *and* JOHN H. SHENK, *Urbana, Illinois*

Chemistry Department, University of Illinois

IT HAS long been known that formaldehyde produces very marked changes in the properties of proteins, principally that of hardening, but no explanation has been found as to the nature of the reaction. Of course, one would expect formaldehyde to attack first the free amino groups, and if further reaction takes place, it would have to attack the amide linkage, probably bridging across from one protein chain to another. This would tie the chains very tightly together and would account for the relatively large increase in weight when proteins are treated with formaldehyde, and also would probably make the protein harder.

When dry gelatin was treated with formalin, it became very gelatinous. When this was left to dry in air, it became quite tough and finally dried quite hard. Dry gelatin gives an x-ray diffraction pattern which is very similar to that of air-dried egg albumin, but after treating with formaldehyde the pattern has been greatly changed, as shown in Figure 1. About five hours are required to obtain a fair pattern of the air-dried protein, while the principal spacing shown in the formaldehyde-treated protein will appear in one-half hour. The outstanding thing about this pattern is the strong ring representing an interplanar spacing of 3.9 Å.U.

The fact that the 3.9 Å.U. spacing gives such a strong interference shows that the treatment of a protein with formaldehyde causes a much more regular alignment of the polypeptide chains. The spacings in Å.U. appearing on this pattern are as follows:[1] 12.5 w, 6.67 w, 4.79 w, 3.9 m, beta, 3.9 vs, 2.61 vw beta, 2.79 w, and 2.6 bs. The two strong interferences at 3.9 and 2.6 appear on the diffraction patterns of all of the proteins attacked by formaldehyde, such as egg albumin and hemoglobin. The fibrous proteins—hair, silk, tendon, and feathers—when soaked in formaldehyde give exactly the same diffraction patterns as before treatment, but if they are first swollen and then treated with formaldehyde while in that condition, they give the same strong rings as gelatin, although these may be fibered.

Zein, a prolamine, is insoluble in water and is not strongly attacked by formaldehyde as can be seen in Figure 3, although the 3.9 ring does appear. The two protein rings also show very plainly. The zein is more readily attacked if it is swollen in 50 per cent alcohol before treating with formalin. Swelling with 50 per cent alcohol and then drying does not change the pattern of the protein (Fig. 2), but the treatment of the swollen zein with formalin increases the intensity of the 3.9 Å.U. ring and also brings out the 2.6 Å.U. ring which is not visible in the pattern obtained by treating dry zein with formalin (Fig. 4).

In the study of the action of formaldehyde on fibrous proteins, the following substances were used: hair, silk, feathers, and tendon. All were extracted with ether to remove lipoid substances which would prevent the penetration of the formalin solution. The specimen-to-film distance was 5 cm. for tendon and feathers, and 3 cm. for hair and silk. The x-rays used were from a copper target and were filtered through nickel foil. In every case, the x-ray beam was transmitted perpendicularly to the fiber axis. Treatment of the dry fibers with formalin does not change the x-ray pattern.

Dry, ether-extracted tendon swells very readily in both 0.1 N and N potassium hy-

[1] Intensities are indicated by: w, weak; m, medium; vs, very strong; vw, very weak; s, strong.

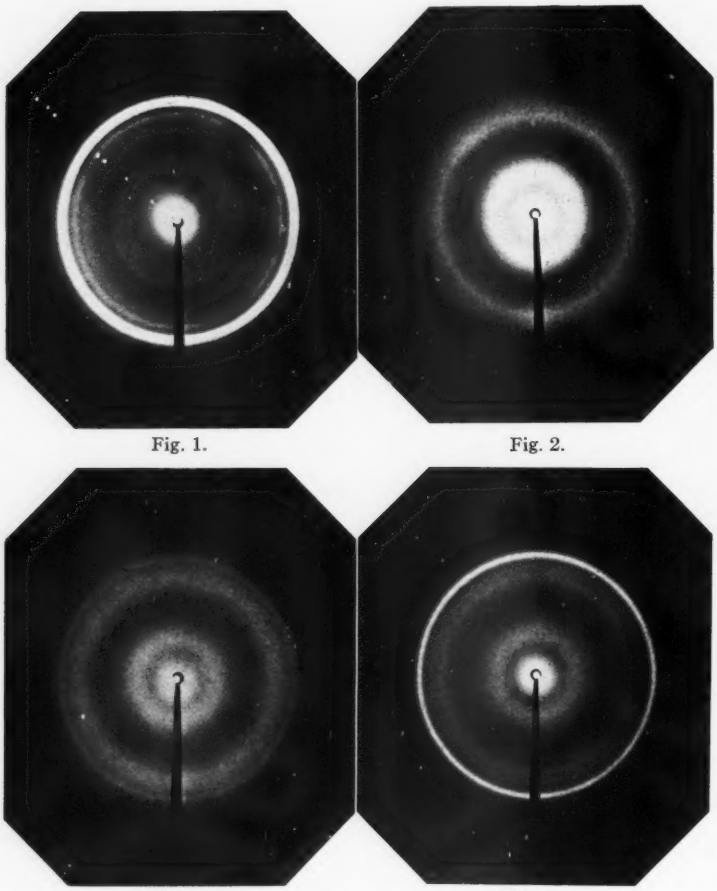


Fig. 1. Fig. 2.

Fig. 3. Fig. 4.

Fig. 1. Gelatin treated with formaldehyde.
Fig. 2. Zein + 50 per cent alcohol, air-dried.
Fig. 3. Dry zein + formaldehyde.
Fig. 4. Zein + 50 per cent alcohol + formaldehyde.

droxide; however, prolonged treatment in N KOH causes disintegration of the fiber. A fiber was swollen in 0.1 N KOH and divided into two portions, one of which was removed from the solution and air-dried while the other portion was placed in formalin for several hours and then air-dried, both under slight tension. Diffraction patterns of the untreated and the two treated samples are shown in Figures 5–7. The untreated tendon gives a well oriented pattern which shows some long spacings. Swelling has little effect, excepting the disappearance of the long spacings, and these would appear if dried under sufficient tension. Although formalin has no effect upon the dry fiber, it readily attacks the swollen tendon, causing the appearance of the 3.9 and 2.6 Å.U. spacings so outstanding on the formalin-treated gelatin pattern. Fibering is still evident and the fibering of these two spacings is 90 degrees apart.

Feather quills were similarly treated. The original fiber pattern is not changed much by swelling in sodium hydroxide solution and then drying. Formaldehyde again brought out these two strong rings fibered perpendicularly to each other, although most of the fiber pattern of the original feather still appears. The feather swelled rather

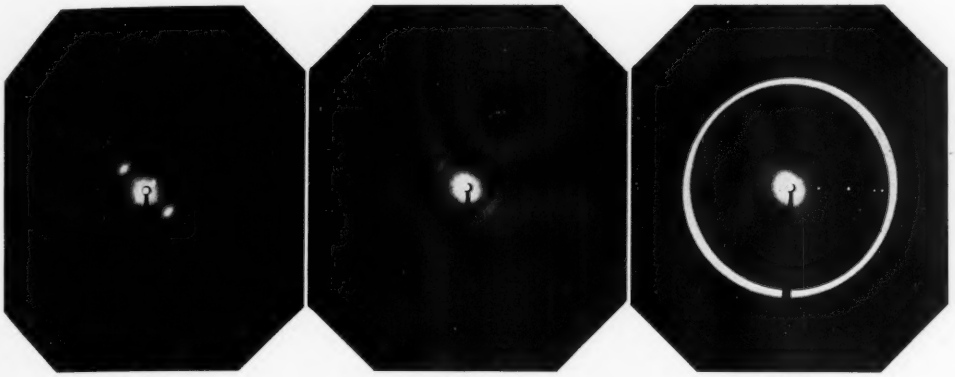


Fig. 5. Fig. 6. Fig. 7.

Fig. 5. Tendon, not treated.
Fig. 6. Tendon + 0.1 N KOH, air-dried.
Fig. 7. Tendon + 0.1 N KOH + formaldehyde.

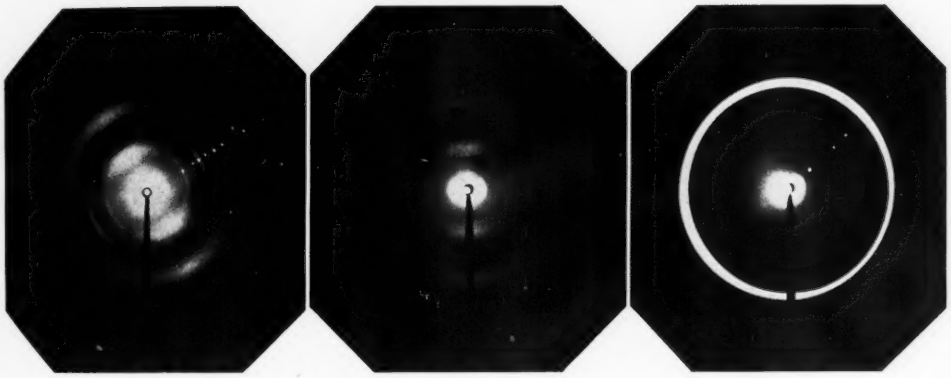


Fig. 8. Fig. 9. Fig. 10.

Fig. 8. Feather, untreated.
Fig. 9. Feather + NaOH.
Fig. 10. Feather + NaOH + formaldehyde.

rapidly in N KOH, but soon lost much of its tensile strength and when treated with formalin, the diffraction pattern showed practically no fibering. The feather patterns are shown in Figures 8–10. Although hair does not swell much in the alkaline solution used, it gives the same diffraction rings when treated with formalin. A pattern of untreated hair is shown with the formalin-treated swollen hair in Figures 11 and 12. The two main rings again show fibering at 90 degrees.

Silk does not swell readily in acid or alkaline solutions and, therefore, one would not expect formaldehyde to have much action on it. This is found to be the case as shown in Figures 13 and 14. The only difference noted in the two patterns is a slightly better oriented pattern than shown by the treated sample.

An examination of the gelatin pattern would lead one to believe that the two strong rings are second and third orders of a 7.8 Å.U. spacing, but when one compares the gelatin pattern with that of formalin-treated fiber, it is at once evident that these two rings are produced by two entirely different spacings because they are fibered perpendicularly to each other. If they were produced by the same spacing, they would be fibered in the same direction. The amino acid residue length in natu-

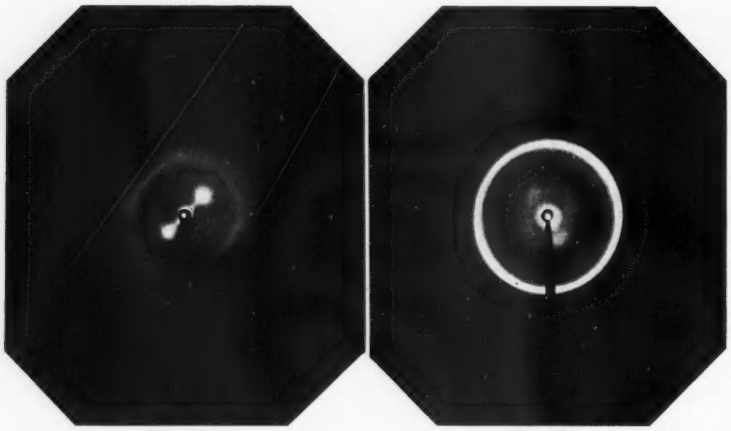


Fig. 11. Fig. 12.

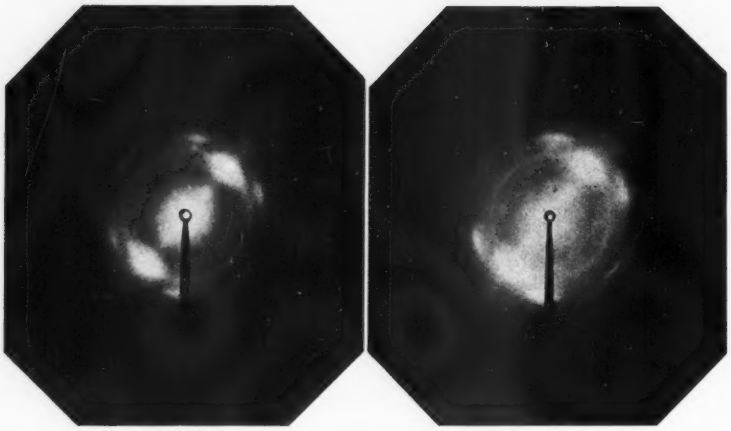


Fig. 13. Fig. 14.

Fig. 11. Normal hair, untreated.
Fig. 12. Hair + N NaOH + formaldehyde.
Fig. 13. Silk.
Fig. 14. Silk + N KOH + formaldehyde.

rally occurring silk and the keratins is about 3.5 Å.U., the distance from one nitrogen atom in the polypeptide chain to another. If formaldehyde reacts with the amide nitrogens of two neighboring chains, we should expect a strong ring spacing at 3.5 Å.U. Since a strong ring is found at 3.9 Å.U., the reaction of the protein with formaldehyde causes a lengthening of 0.4 Å.U. per amino acid of the chain. The 2.6 Å.U. spacing is probably the second order of an increased "backbone" spacing due to the reaction. The 12.5 Å.U. ring represents the silver absorption edge due to the 3.9 Å.U. spacing and the 6.67 ring represents the bromine absorption edge.

Swelling of the fibers pushes the protein grids farther apart, thereby facilitating the penetration of the formaldehyde solution so that it could react—and may also cause a certain amount of hydrolysis, which was undoubtedly the case when N KOH solution was used for swelling tendon and feather fibers. Silk fibroin alone is unaffected by formaldehyde; of all the proteins, it alone has hydrogen atoms as the R groups on the polypeptide chains.

It seems logical to assume that the reaction of formaldehyde on proteins takes place at the amide nitrogen because all of the proteins except silk show similar diffraction effects when treated with formalin. Because of the extreme intensity of the 3.9 Å.U. ring, there has to be a very definite alignment of a great number of atoms or chains which could only be due to the placing of a large number of atoms close together at regular intervals. Also, since all of the proteins give the same rings in the same positions with the same relative intensities, the reaction is very closely connected with the fundamental structure of the molecule.

SUMMARY

1. Proteins such as gelatin, egg albumin, and hemoglobin after treatment with formaldehyde give diffraction patterns containing two strong rings, neither of which appears in the pattern of the original protein, and both are very much stronger than the original pattern.

2. Dry zein is not greatly affected by formaldehyde but if swollen in 50 per cent alcohol, it is more readily attacked as shown by the intensity of the rings on the diffraction patterns.

3. Fibered proteins as hair, tendon, and feather quills are not attacked enough by formaldehyde to change the diffraction pattern, but if swollen in an alkaline solution and then treated with formaldehyde, the diffraction pattern resembles that of the formalin-treated gelatin except that fibering still appears and the strong rings are fibered 90 degrees apart. There is no effect on silk fibroin.

4. It is believed that the formaldehyde reaction takes place on the amide nitrogens, tying two adjacent chains together.

5. The 3.9 Å.U. spacing is probably due to the slightly extended amino acid residue and the 2.6 Å.U. spacing is probably the second order of an increased "backbone" spacing.

NEOPLASMS INVOLVING THE DUODENUM[1]

By SYDNEY WEINTRAUB, M.D., *and* ALLAN TUGGLE, M.D., *New York City*

From the Department of Radiology, Cornell-New York Hospital Association

NEOPLASMS occurring in the region of the duodenum, *i.e.*, those arising either in the gall bladder, bile ducts, head of the pancreas, or the duodenum itself, present unusual difficulties in diagnosis, not only to the clinician, but also to the roentgenologist. Although the patient may have a well advanced carcinoma in this region the x-ray evidence of a lesion may be easily overlooked unless a minute study of the pattern and contour of the entire duodenum is made. In this study 20 cases have been reviewed, in all of which the diagnosis has been confirmed either by operation, autopsy, or biopsy. The lesions proved to be as follows:

Carcinomas of the head of the pancreas	8
Lymphoblastomas (lymphosarcoma or Hodgkin's of the regional lymph nodes)	4
Carcinomas of the gall bladder and bile ducts	3
Carcinomas of the ampulla of Vater	2
Primary carcinomas of the duodenum	2
Leiomyosarcoma of the pancreas	1

There was definite x-ray evidence of a lesion in 18 of these cases. Of the two cases in which the radiologic examination was reported as negative, one proved to be a tumor 2.5 cm. in diameter involving the duct of Wirsung, concerning which the pathologist reported as follows: "The stomach and duodenum were opened *in situ* and the mucous membrane was intact." In the other case an angulation of the second portion of the duodenum suggesting adhesions was reported, but the autopsy revealed a carcinoma at the head of the pancreas involving the gall bladder and regional lymph nodes. The pathologist, however, was not able to identify the primary site of the tumor.

In the 18 cases in which there was definite x-ray evidence of an abnormality, it manifested itself by one or several of the following signs:

(1) Obstruction of some portion of the duodenum;

(2) Filling defect of the duodenum;

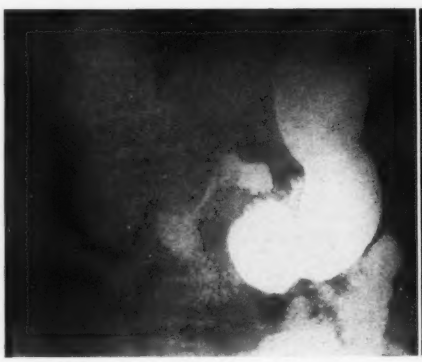


Fig. 1.

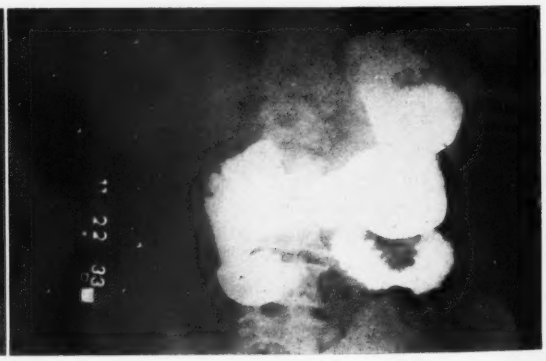


Fig. 2.

Fig. 1. Carcinoma of the gall bladder. Constant pressure defect on the upper portion of the second duodenum.

Fig. 2. Lymphoblastoma of the pre-pancreatic glands. Marked obstruction of the third duodenum, with involvement of the proximal jejunum.

(3) Disturbance of the mucosal pattern;

(4) Pressure defects of the bulb;

[1] Presented before the Radiological Society of North America at the Twenty-second Annual Meeting, at Cincinnati, Nov. 30–Dec. 4, 1936.

(5) Distortion of the normal duodenal swing;
(6) Pressure defects on the greater curvature of the antrum;
(7) Air in the bile ducts.

In the four cases in which the duodenum was primarily involved (ampullary and primary duodenal carcinomas), the diagnosis was comparatively easy. There were filling defects either in the second or third duodenum, with varying degrees of obstruction, and the normal pattern of the mucous membrane was destroyed. In one case of ampullary carcinoma the lesion was first suspected when air in the bile ducts was visualized on the films of a Graham test. The subsequent examination of the duodenum revealed a fistulous communication between the duodenum and the biliary tract. We believe that air in the bile ducts is significant and a neoplasm should be suspected.

Fig. 3. Carcinoma of the head of the pancreas. Note pressure defect on the greater curvature of the pyloric antrum and constant defect of the second duodenum, together with a fistulous tract.

The carcinomas of the biliary system are more apt to give pressure defects with or without obstruction to the duodenum, but they do not produce disturbances in the mucosal pattern. The defect is usually noted in the bulb or at the beginning of the second duodenum. It is not uncommon to find gallstones in carcinomas of the gall bladder. The lymphoblastomas found in the regional nodes at the head of the pancreas also produce defects, particularly at the pyloric antrum. They may cause duodenal obstruction, but as a rule do not destroy the mucosal pattern unless the walls of the duodenum have been invaded.

In the carcinomas at the head of the pancreas no one finding was constant. They showed signs of obstruction, pressure defects, destruction of the mucosal pattern,

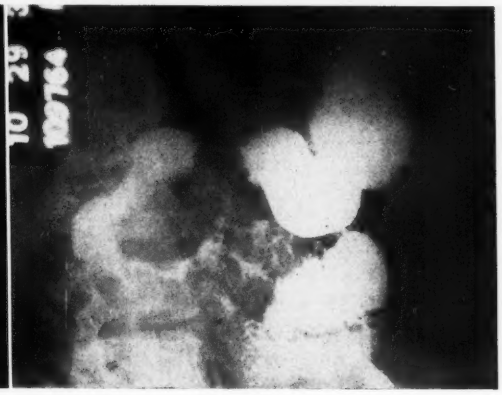


Fig. 4. Fig. 5.

Fig. 4. Carcinoma of the ampulla of Vater. Note filling defect and narrowing of the second duodenum.
Fig. 5. Primary carcinoma of the duodenum. Obstruction of the second duodenum and destruction of the mucosal pattern of the third duodenum.

distortion of the duodenal loop, or a combination of these findings. In cases in which the tumor is so small or so located that it does not impinge upon the surrounding structures, the diagnosis cannot be made radiographically. The normal swing of the duodenum was distorted in some of our cases but in not one of them was it uniformly widened, as one is apt to see in cysts of the pancreas. The leiomyosarcoma of the pancreas showed a crater in the second duodenum. This was called an ulcer in another institution one year after our original diagnosis of carcinoma. We have seen several such cases in which the lesion simulated an ulcer of the second duodenum, but the patient eventually succumbed to a neoplasm. It may be of some significance to note that six out of the eight carcinomas of the head of the pancreas showed a lumbar scoliosis away from the affected side. In the other types of lesions this sign was not constant.

A brief résumé of the clinical data shows that there were 12 females and 8 males. The symptoms were usually of short duration and 14 patients were complaining for only three months or less. The loss of weight and strength was rapid, and when there was obstruction to the duodenum these symptoms became very marked because of the persistent vomiting. Jaundice was present in 12 cases and a mass was palpable in 8. Out of 15 cases in which the stool was examined for blood, 9 showed a positive benzidine reaction.

We are also impressed by the fact that the radiologist can be of help in more definitely localizing some of these lesions. Many of these become quite extensive and involve most of the structures in the right upper quadrant. On the operating table, at times, it is difficult to determine the primary site of the tumor and even the pathologist may be uncertain. In some of our cases the lesion was reported to be situated at the ampulla of Vater, but the radiographs presented a normal second duodenum and a filling defect in the third portion. The ampulla of Vater, according to the anatomist, enters the duodenum at the upper third of the second portion, 7.5 to 10 cm. from the pylorus. Unless the duodenum is incised, one cannot be certain that the carcinoma is ampullary, and we believe that some of these are really primary carcinomas of the duodenum.

CONCLUSIONS

The x-ray diagnosis of neoplasms in the region of the pancreas, biliary system, and duodenum is difficult.

Out of the 20 cases studied, 18 showed x-ray evidence of a lesion.

A minute study of the contour and mucosal pattern of the entire duodenum should be a part of the routine procedure in every gastro-intestinal series.

DISCUSSION

DR. RABUN T. WILSON (Austin, Texas): The rarity of a disease serves to intensify our interest in its investigation. Often a study leads to the discovery that it occurs more frequently than has heretofore been believed. While this is true with the particular subject in hand, it nevertheless remains a comparatively rare condition and one of unusual interest.

Lesions in the duodenum are quite common, occurring in approximately 10 per cent of gastro-intestinal studies and constituting 80 per cent of organic lesions in the gastro-intestinal tract, but all of these, in my own experience, with one exception, have been of the benign ulcerative type.

A malignant growth from the stomach or other neighboring structures, invading the duodenum by extension, is a common finding but a primary ulcerative lesion in the duodenum is rare.

My experience is confined to the one case out of approximately 25,000 gastro-intestinal studies. This lesion occurred in a man 55 years of age, was located near the apex of the bulbar or first portion of the duodenum, and was producing well-marked luminar reduction and a well-defined mucosal irregularity with almost complete obstruction resulting. Its malignant nature was not recognized roent-

genologically but was determined by microscopic studies after operation.

A few years ago in a symposium on peptic ulcer on a State Medical Society program, in presenting the roentgenologic phase of the subject I showed slides of this case. In the informal discussion which followed, the Professor of Medicine in a medical school expressed his surprise, stating that he was not aware that primary malignancy of the duodenum had ever been recognized. A search of the literature at that time revealed only a few cases, even from the large clinics, but more recently other reports have been added.

All of us have encountered cases arising in the ampulla of Vater, the head of the pancreas, the liver, and bile ducts. Several such cases have come under my observation, though the records at this time are not available.

The roentgenologic diagnosis of these cases is difficult because there is often such complete obstruction or so little mucosal change that the evidence is not characteristic. Even the pathologist, as has been stated by the author, is often unable to trace accurately the origin of a disease in the mass of malignant tissue.

The authors of the paper just read are to be congratulated on finding diagnostic x-ray evidence of the lesion in such a high percentage of their cases. This paper and other literature contain reports of malignancies of both carcinoma and sarcoma type, but the same difficulties in roentgenologic recognition are encountered with both lesions, since the evidence is confined to the mucosal and luminar changes, together with resulting functional disturbances.

Theoretically, at least, one would give preference to carcinoma when mucosal changes predominate. Pressure defects of the duodenum or antrum of the stomach, or a distortion of the normal duodenal swing would appear to be rather remote evidence upon which to base a diagnosis of this condition, since these may occur in tumors arising in so many neighboring structures.

It would appear, therefore, that the principal evidence would depend upon the authors' signs 1, 2, and 3, namely, obstruction of some portion of the duodenum, filling defect of the duodenum, and disturbances of the mucosal pattern.

This discussion will doubtless serve to stimulate interest in this field and may result in the conclusion that the condition is more frequently encountered than has heretofore been believed.

Careful evaluation of the authors' signs may also help us in differentiating the benign from the malignant neoplasms in this region.

DR. AUGUSTUS W. CRANE (Kalamazoo, Mich.): I was interested in what Dr. Wilson said—that primary carcinoma of the duodenum is exceedingly rare. During my lifetime I have seen one case. That was located in the second ascending limb of the duodenum.

The part which interested me particularly in that case was the behavior of the stomach in the presence of a carcinoma of the duodenum, and to be compared with the behavior of the stomach was an ulcer in the same locality. It happened that at the time of this carcinoma of the duodenum, we had also an ulcer of the duodenum in the same locality. Both were operated upon and the nature of the trouble was undoubted.

In the case of the carcinoma of the duodenum, the only evidence we had of some involvement of the bowel, of the intestine, was the fact that there was a 48-hour retention of the stomach contents without an obstruction of any kind to be demonstrated. At times we imagined we saw an antiperistalsis but at least there was no obstruction shown at operation and yet there was this massive gastric residue.

I offer this as one of the signs of an involvement of the intestine which may not otherwise be identified.

This case of carcinoma of the duodenum was operated upon at the Mayo Clinic finally and no obstruction was found. It was a "napkin ring carcinoma" of the duo-

denum, and the patient finally died from a metastatic involvement of the other organs. The postmortem revealed the true situation.

Dr. Charles Mayo wrote me a very full letter about it, and the fact of the absence of an obstruction is what I want to impress upon you—that you can have massive gastric residue in those cases with absolutely no obstruction.

As a comparison with what happens in the case of ulcer of the duodenum, you have exactly the same reaction of the stomach as when the ulcer is located in the bulb of the duodenum—that is, a rapid peristalsis with rapid gastric emptying. The expulsion of gastric contents goes on so rapidly that you would naturally suspect that you had the familiar lesion in the duodenal bulb.

I might say also, in regard to the rarity of these lesions, that at this time Dr. Carman was living. He had also examined this same patient after the latter had been sent to the Clinic for operation. He told me, as I recall, that in some 30,000 cases of cancer at the Mayo Clinic they had had only four cases of primary carcinoma of the duodenum.

DR. TUGGLE (closing): It is our plea that in patients suspected of having carcinoma in the right upper quadrant, a very minute study of the entire duodenum be made. You will be surprised if you will go back over your old films of proven cases of carcinoma in this region and note the smaller changes in the mucosal pattern that you will find.

Primary carcinoma of the duodenum is rare. Out of our cases there were four—that is, two ampullary and two primary duodenal.

VANISHING LUNGS: A CASE REPORT OF BULLOUS EMPHYSEMA

By RICHARD M. BURKE, M.D., Veterans' Hospital, *Sulphur, Oklahoma*

BULLOUS emphysema is usually associated with strictures of the bronchi. These narrowed bronchi permit the inspired air to enter the alveoli, but on expiration the outlet closes and back-pressure is created, a mechanism which in time causes dilatation, atrophy, and rupture of the alveoli. Finally, large emphysematous bullæ evolve from the coalescing alveoli. The histopathology of blebs and bullæ has been described by W. S. Miller (1, 2).

The etiology of the obstruction in bullous emphysema is frequently obscure. Presumably in some instances the bronchial changes are the result of developmental errors; in others, pulmonary fibrosis of unknown etiology such as described by Hampton (3) may be the cause. The best understood type is that in which bullæ follow localized productive changes such as result from tuberculosis. Haymaker and Karan (4), and Freedman (5) have reported examples of the latter type.

The diagnosis, made largely from the x-ray examination, may be confused with partial pneumothorax, cystic disease, and tuberculous cavitation. The x-ray appearance has been discussed by Haahti (6), Vadone and Maissa (7), Freedman (5), and others.

The following, I believe, is one of the most advanced cases of bullous emphysema recorded.

CASE REPORT

In 1929, I. J. S., 28 years of age, was forced to quit work. His complaints then were general weakness and increasing shortness of breath. A diagnosis of tachycardia, of unknown etiology, and chronic bronchitis was made by the Veterans Bureau. According to the history, he had spent four years in the Army where his duties had been light. After his discharge in 1923, he worked as a day laborer, spending several years as an oil field worker.

In 1930 he entered the Veterans Hospital, and a diagnosis of bilateral pneumothorax, of unknown etiology, was recorded. Following this, he visited several other hospitals and also returned here on three occasions. His medical file reveals that he was somewhat of a diagnostic problem wherever he went. One diagnosis given was congenital atelectasis of both upper lobes. However, this case was discussed and correctly diagnosed in a paper by Korol and Ensign (8) in 1934.

His last admission here was on Oct. 4, 1935, at which time he was quite dyspneic, weak, and moderately emaciated. The patient's past history was negative except for typhoid fever in 1913. Also he related that as a child he tended to tire rather easily.

Physical examination was essentially negative except for the chest. Blood pressure 108/96. Rate rapid with tones weak in character. Chest was of the emphysematous type. Hyper-resonant on both sides. Breath sounds absent except over the bases. Whispered voice practically absent. Coin test negative. No suggestion of amphoric quality to breath sounds. Diagnostic pneumothorax gave neutral readings on both sides.

The patient's chest films, taken at intervals during past six years, were reviewed. They revealed a progressive bilateral downward loss of lung markings. Stereoscopic views showed fine web-like markings extending upward from the areas of normal appearing lung.

Figures 1 to 6 illustrate how the lungs seemed to vanish. A film taken on July 28, 1930, shows the pulmonary markings absent down to the third rib on the right and the fourth rib on the left (Fig. 1). A postmortem film (Fig. 6) shows absent markings to the fifth rib on the right and to the eighth rib on the left.

In 1935 a circumscribed mass was first

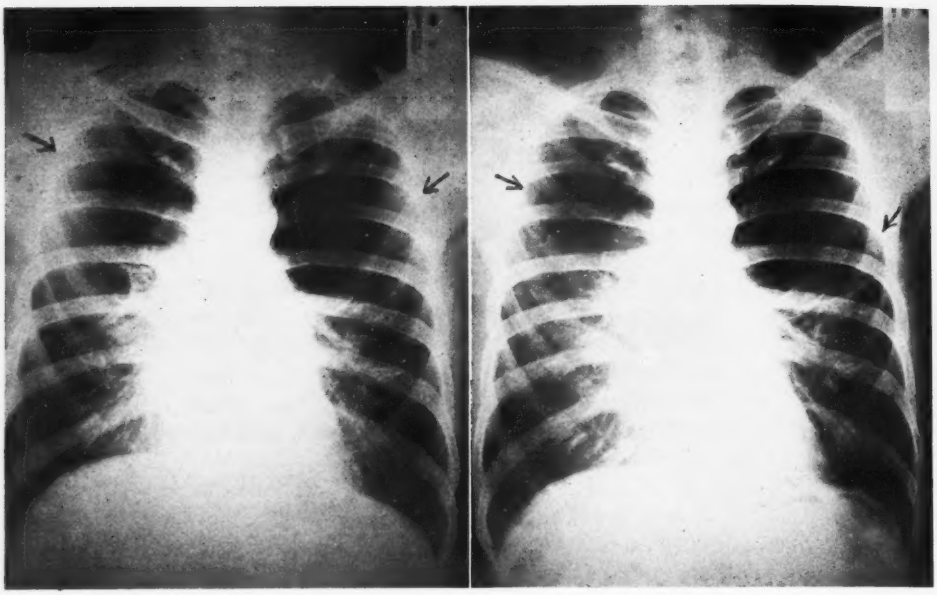


Fig. 1. Fig. 2.

Fig. 1. Bilateral bullous emphysema. More pronounced on the left. Figures 1 to 6 show its progression. Lung markings absent down to the levels indicated by the arrows (July 28, 1930).
Fig. 2. Appearance seven months later (Feb. 23, 1931).

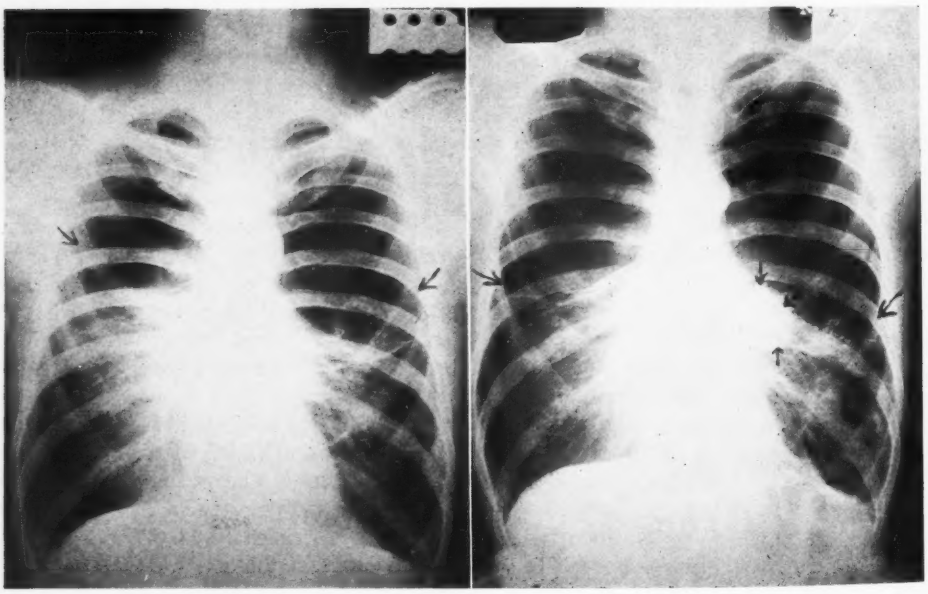


Fig. 3. Fig. 4.

Fig. 3. The downward recession of lung markings continues (Nov. 7, 1932).
Fig. 4. Circumscribed mass appears in left hilus region (Oct. 4, 1935).

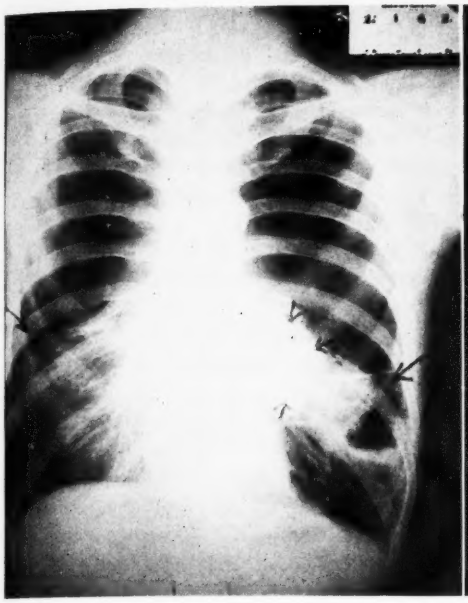


Fig. 5.

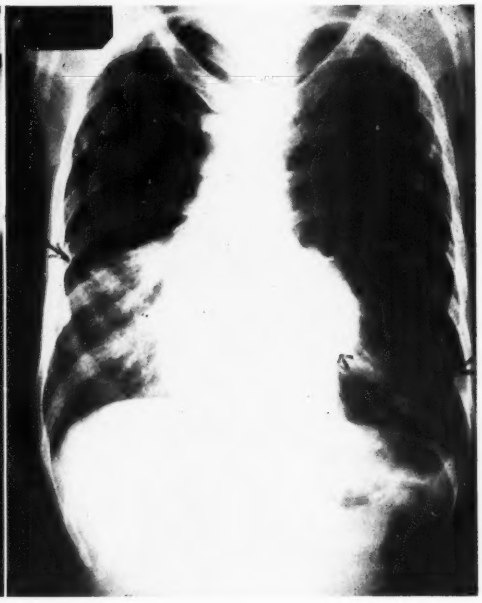


Fig. 6.

Fig. 5. Lung tissue continues to fade. Mass in left hilus increases in size (Feb. 19, 1936).
Fig. 6. Postmortem film. Mediastinal shift to the right is seen. Practically no lung tissue visualized on the left while about half of the right lung remains (April 4, 1936).

noted in the left hilus region (Fig. 4). This slowly increased in size until death.

The patient was subject to acute attacks of dyspnea, suggesting a cardiac asthma rather than a typical bronchial asthma. His respirations would become rapid and shallow during an attack, and he often would request the windows to be opened. He complained of tightness in the chest. These attacks started in 1932 and were at first partially relieved by ephedrine and adrenalin. Later they became more severe and nothing short of morphine would relieve him. During the last few months the attacks occurred five or six times a week and usually lasted from 30 to 60 minutes. Between attacks he was relatively comfortable, even up to the time of his death.

He became more emaciated, developed an exhaustion physchosis, and died on April 5, 1936.

An autopsy was done. Upon opening the chest, the lungs ballooned out and overfilled the chest cavity as though under pressure. This was especially pronounced on the left (Fig. 7). The upper lobes remained distended even after considerable manipulation. Both lungs had a lobulated appearance with many blebs seen.

The right upper lobe contained one large bulla plus a number of lesser emphysematous bullæ and blebs about its lower margin. No gross communication between the various bullæ could be demonstrated.

Fig. 7. View of chest upon removing ribs. Shows over-distended appearance of upper lobes. On opening these lobes practically empty pleural sacs were revealed.

The upper left lobe was adherent to the chest wall and mediastinum. Practically all that remained of this lobe was an

Fig. 8. The giant bulla which occupied most of the left pleural cavity.

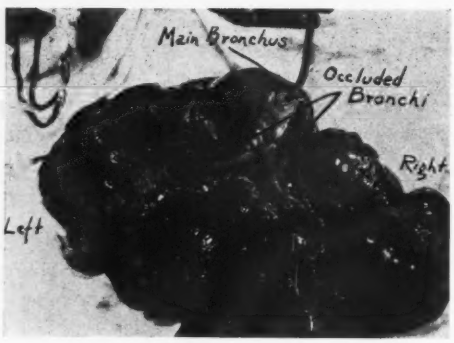


Fig. 9. Posterior view of lungs. The occluded upper lobe bronchi are indicated.

empty pleural sac (Fig. 8). This giant bulla occupied four-fifths of the left chest cavity.

Analysis of gas samples taken from these bullæ showed atmospheric air. (It has been suggested that the acute dyspnea in these cases was due to rebreathing air from these large sacs which were thought to contain a high percentage of CO_2.)

Section of the compressed lower lobes revealed some increase in fibrosis but no evidence of tuberculosis. A solid tumor, 4 cm. in diameter, was found along the left main descending bronchus. Tissue examination showed it to be a bronchiogenic carcinoma, Grade IV.

Dissection of the bronchial tree showed narrowing and occlusion of bronchi going to the upper lobes (Fig. 9). Why this narrowing and occlusion had occurred was not apparent. The increase in fibrosis which was present would hardly explain it. No definite openings into the large bullæ could be demonstrated. Presumably a communication did exist but was closed off some time prior to death.

The remainder of the autopsy revealed no other significant findings.

DIAGNOSIS

There are a few points in diagnosis which might be recalled. There was little cough or sputum. Unexplained progressive increasing shortness of breath was the most significant symptom, plus periodic spells of dyspnea in the advanced stages. The latter is thought to have been due to pressure on the mediastinum by the distended lungs. Also pulmonary hypertension probably developed, due to the diminishing lung tissue and fibrosis.

Although physical signs suggested pneumothorax, the amphoric quality of the breath sounds sometimes heard in pneumothorax was absent.

On x-ray examination, a gradual fading of the lung markings, usually beginning at the apices, was noted, to be differentiated from pneumothorax by the absence of a sharp lung line. Close examination showed fine unanatomical markings in the radiolucent areas, best seen on stereoscopic films, markings which were due to fibrous septa—all that remained of the lung tissue. In pneumothorax the affected area regresses, while in emphysema it progresses. Also, a so-called chronic pneumothorax will invariably lead to fluid formation.

A large bulla can simulate a tuberculous cavity. We have seen one case and Miller (9) has described such a case. A differential point in such instances is mentioned by Freedman (5), who states that a number of curved shadows around the area of diminished density should arouse one's suspicions that they represent something else besides cavitation.

Cystic disease of the lung usually presents circumscribed areas of increased brilliancy. Fluid levels are not uncommon. The walls of the cyst are well defined in contrast to the delicate filmy margins of bullæ and blebs.

Lipiodol will often visualize the stenosed or occluded bronchi. Serial films showing the progress of the emphysema are pathognomic. Films taken in the knee-chest position have been recommended, a procedure which is said to cause congestion of the blood vessels in the upper lung-fields and thus enable them to be visualized. In the above case we were unable to note any change following such a procedure. Fluoroscopy will show a slight increase in the size of the bullæ on expiration as compared with inspiration. Diagnostic pneumothorax ordinarily should be attempted only when other methods fail.

As far as we know the associated bronchiogenic carcinoma did not play any part in the evolution of the bullæ we have described. Swenson (10) and Freedman (5) have reported primary malignancy associated with bullous emphysema.

Treatment.—The establishment of outside fistulæ and extirpation of the affected areas have been attempted. Removal of air will sometimes give temporary relief.

CONCLUSION

A case of bilateral bullous emphysema with an associated bronchiogenic carcinoma is reported in which two-thirds of the lung tissue had been replaced by giant bullæ.

BIBLIOGRAPHY

(1) MILLER, W. S.: A Study of the Human Pleura Pulmonalis: Its Relation to the Blebs and Bullæ of Emphysema. Am. Jour. Roentgenol. and Rad. Ther., 1926, **25,** 399.

(2) Idem: A Further Study of Emphysematous Blebs. Am. Jour. Roentgenol. and Rad. Ther., 1927, **18,** 43.

(3) HAMPTON, A. O.: Progressive Idiopathic Pulmonary Fibrosis Associated with Emphysema. New Eng. Jour. Med., Dec. 12, 1935, **213,** 1174–1177.

(4) HAYMAKER, W., and KARAN, A. A.: Giant Saccular Bulla of Lung: Report of Case with Discussion of its Formation. Am. Rev. Tuberc., February, 1935, **31,** 240–249.

(5) FREEDMAN, E.: Emphysematous Blebs and Bullæ. Am. Jour. Roentgenol. and Rad. Ther., March, 1936, **35,** 324–333.

(6) HAAHTI, H. G.: Beobachtungen ueber einen Fall mit ausgedehnten intrapulmonalen lufthaltigen Hohlraumen. Acta Radiol., 1932.

(7) VADONE, A., and MAISSA, P. A.: Giant Vesicular Emphysema as Cause of Round Shadows in Roentgen Picture of Lung. Prensa méd. argent., June 7, 1933, **20,** 1254–1256.

(8) KOROL, EPHRAIM, and ENSIGN, C. F.: Bullous Emphysema (?) or Bilateral Pneumothorax (?). RADIOLOGY, August, 1934, **23,** 223–227.

(9) MILLER, W. S.: A Tuberculous Lung in which a Large Emphysematous Bulla was Mistaken for a Cavity. Am. Rev. Tuberc., September, 1933, **28,** 359–369.

(10) SWENSON, P. C.: Personal communication.

EDITORIAL

LEON J. MENVILLE, M.D., *Editor* HOWARD P. DOUB, M.D., *Associate Editor*

GASTROSCOPY AND THE RADIOLOGIST

In this country, the endoscopic examination of the stomach, commonly called gastroscopy, is a relatively new procedure. The numerous contributions of Dr. Rudolph Schindler, one of the pioneers in this field, and his students are, no doubt, largely responsible for the present interest in this method of examination. Such an interest among radiologists was well demonstrated by the attention directed to the Symposium on Gastroscopy and Roentgenology in the Diagnosis of Diseases of the Stomach at the recent meeting of the Radiological Society of North America. The papers in this symposium, which will be published in a forthcoming issue of RADIOLOGY, will, unquestionably, be widely read.

Radiologists, particularly, are giving consideration to this diagnostic procedure because of the possible effect upon their practice. Any non-hazardous means, furthering our knowledge of gastric diseases, is welcome. The direct inspection of the mucous membrane of the stomach, which this procedure affords, offers many opportunities for the intimate study of gastric lesions. While not entirely without danger—occasional perforations may occur—examination with the Wolf-Schindler flexible gastroscope is not exceedingly difficult or trying to the patient. The effect which the widespread application of this method may have upon radiological practice lends itself to the following three considerations.

First of all, it should be clearly understood that the gastroscopic examination supplements rather than competes with the roentgen examination of the stomach. An adequate roentgen study is an absolute prerequisite for an intelligent, conservative endoscopic examination. The practice of gastroscopy, in any community, should stimulate an additional interest in diseases of the stomach. Hence, it seems probable that the introduction of this procedure will increase rather than decrease the number of gastro-intestinal cases referred to the radiologist.

Secondly, gastroscopy creates a new stimulus and a new guide for the activities of the roentgen diagnostician. At one time surgery was the chief factor in the discipline, so necessary to keep us from degenerating into smug complacency. The decline of surgical intervention in non-tumorous lesions of the stomach, together with the realization that inspection and palpation of the exterior of the stomach gives no final assurance of the presence or nature of a lesion have somewhat modified the salutary effects of laparotomy for determining the value of roentgen conclusions. The development of another method of examination should give the radiologist another desirable control for the accuracy of his findings.

Finally, and to the radiologist, most important of all, the endoscopic study of the stomach may prove to be an educational force of considerable importance. The unsatisfactory character of the usual gross and histological investigations of the pathology of gastric diseases is well known. The results, at this date, indicate that longer experience with direct inspection of the gastric mucosa in the living individual will teach us many facts about those diseases, particularly gastritis, which, prior to this time, have been difficult to elucidate. The gastroscopic observation of mucosal erosions, gastric ulcers, and the various types of gastric tumors should prove of great value in establishing anatomical data to guide us in the interpretation of roentgen observations.

Although realizing that it is a supplementary procedure, that it has many sharp limitations and that it is not without danger, the radiologist should welcome and encourage the establishment of intelligent and conservative gastroscopy in his community.

LEO G. RIGLER, M.D.

ANNOUNCEMENTS

EXCEPTIONAL EDUCATIONAL OPPORTUNITIES AT THE FIFTH INTERNATIONAL CONGRESS OF RADIOLOGY

An unusual opportunity will be offered at the Fifth International Congress of Radiology, Sept. 13 to 17, 1937, Palmer House, Chicago, Illinois, for instruction in certain selected fields of radiology. The courses to be offered are as follows:

ROENTGEN THERAPY IN CANCER

Dr. Henri Coutard, Chief of Roentgen Therapy Service in the Radium Institute, Paris, France.

PROBLEMS IN ROENTGENOLOGICAL DIAGNOSIS

This is an advanced course for experienced radiologists which will deal with difficult problems of diagnosis selected from the vast material available at the Massachusetts General Hospital.

Dr. George W. Holmes, Clinical Professor of Roentgenology, Harvard University Medical School; Director of Radiological Department, Massachusetts General Hospital, Boston, Massachusetts.

Dr. Holmes will be assisted by his associates, Dr. Aubrey O. Hampton, Dr. Richard Schatzki, and Dr. Richard Dresser.

THE FUNDAMENTALS OF ROENTGEN AND RADIUM THERAPY

Prof. Dr. Hermann Holthusen, Professor in the University, Chief of the Radiological Service, St. George Hospital, Hamburg, Germany.

ROENTGEN DIAGNOSIS IN GASTRO-ENTEROLOGY

Dr. B. R. Kirklin, Associate Professor of Radiology, University of Minnesota, Graduate School of Medicine; Chief of the Department of Radiology, Mayo Clinic, Rochester, Minnesota.

Dr. Kirklin will be assisted in this course by other radiologists who are outstanding in this special field.

SPECIAL PROBLEMS OF RADIATION THERAPY RELATING PARTICULARLY TO TREATMENT OF CANCER OF THE CERVIX AND CANCER OF THE BREAST

This course is directed especially to the needs of the specialist in radiology and will be found of particular interest to radiologists located in the smaller centers of population.

Dr. Edwin A. Merritt, Clinical Professor of Radiology, Georgetown University Medical School; Chief Radiologist, Warwick Memorial Clinic and Garfield Memorial Hospital, Washington, D. C.

THE DIAGNOSIS OF BRAIN LESIONS

Dr. Merrill C. Sosman, Assistant Professor of Roentgenology, Harvard University Medical School; Chief of Radiological Service, Peter Bent Brigham Hospital, Boston, Massachusetts.

ELEMENTARY PHYSICS OF RADIATION

Dr. James L. Weatherwax, Philadelphia General Hospital, Philadelphia, Pennsylvania.

Each of the above courses will occupy four hours; that is, one hour on each day of the Congress, September 14 to 17, inclusive. The courses will be given at an hour when they will not conflict with other activities of the Congress, namely, eight to nine in the morning.

Each of the courses will be given in English and will be open to between fifty and one hundred members of the Congress who will register specifically for any one course. Those who have already made application for membership in the Congress may register for one of the courses by writing to the General Secretary, 2561 N. Clark Street, Chicago, Illinois. Applicants for membership in the Congress hereafter may register when they submit their applications. Registrations for the courses will be accepted in the order of their receipt at the Chicago office.

All courses will be given without extra charge to members of the Congress who register for them in the manner indicated above. It is desired that registration be made at as early a date as possible.

NEW APPLICATION FORM

At the order of the Executive Committee of the Radiological Society of North America, a new application form to be used by those applying for membership has been prepared and is now ready for distribution. Please do not use the old blanks which you may have on hand, but secure new blanks from the Secretary-Treasurer, Donald S. Childs, M.D., 607 Medical Arts Bldg., Syracuse, N. Y.

ANNOUNCEMENT OF PRIZE AWARD

A David Anderson-Berry Gold Medal, together with a sum of money amounting to about £100, will be awarded in July, 1938, by the Royal Society of Edinburgh to the person,

who, in the opinion of the Council, has recently produced the best work on the nature of x-rays in their therapeutic effect on human diseases. A similar award will be made every three years.

Applications for this prize are invited. They may be based on both published and unpublished work and should be accompanied by copies of relevant papers.

Applications must be in the hands of the General Secretary, Royal Society of Edinburgh, 22 George Street, Edinburgh 2, by *June 1, 1938.*

AN OPEN LETTER TO RADIOLOGICAL SOCIETIES AND RADIOLOGICAL SECTIONS OF COUNTY AND STATE MEDICAL ASSOCIATIONS

It has been suggested to the Editor that a comprehensive list of such societies would be useful and of interest to the readers of RADIOLOGY, and he is inclined to agree, considering the communications received from time to time from enterprising secretaries. If you belong to such a society, please remind the secretary of it to send information concerning officers and place and time of meeting to RADIOLOGY to be so listed. The Editor can determine from the responses received whether or not there exists a desire for such a listing.

INTERNATIONAL CONFERENCE ON FEVER THERAPY

In conjunction with the International Conference on Fever Therapy to be held at the Waldorf-Astoria Hotel on March 29, 30, and 31, 1937, there will be a scientific and commercial exhibit.

The clinics will be held at the College of Physicians and Surgeons, Columbia University, New York City.

A large attendance of fever therapists from all over the world is expected. A very interesting and instructive program has been arranged and all of those who plan to attend the conference are urged to register promptly with the General Secretary, Dr. William Bierman, 471 Park Avenue, New York City. The registration fee is $15.00.

IN MEMORIAM

The Radiological Society of North America has lost by death two valued members, Dr. Augustus W. Crane and Dr. Thomas O. Menees. In the next issue of RADIOLOGY it is hoped to publish sketches of our late confrères.

BOOK REVIEWS

"RÖNTGENKYMOGRAPHISCHE BEWEGUNG LEHRE INNERER ORGANE" (Roentgen Kymographic Dynamics of the Internal Organs). By VON DR. PLEIKART STUMPF, DR. H. H. WEBER, DR. G. A. WELTZ, VON DR. W. BOHME (Rostock), DR. M. DAHM (Köln), DR. TH. C. NEEFF (Würzburg), DR. G. M. SACK (München-Gladbach), DR. E. SCHOEN (München), DR. G. VON DER WETH (Berlin). A volume of 516 pages, 447 illustrations and explanatory colored plate. Georg Thieme, Leipzig, 1936. Price, 44 RM.

If roentgen kymography becomes an accepted practical method for the examination of the movable organs of the body, this volume will survive as on of the classics of roentgen literature. In any event it will always be a valuable source book for information about the theory and practice of kymography.

Abundantly illustrated, well printed, thoroughly indexed in the table of contents, with a fairly complete bibliography, this book presents the work of Pleikart Stumpf, the pioneer in roentgen kymography, and that of many collaborators covering the entire field of work which the method embraces. Briefly, kymography is a graphic method of portrayed motion. As such, it is applicable to many organs and its usefulness in the study of the heart, pericardium and great vessels, the lungs, diaphragms and thoracic cage, the thyroid gland, the esophagus, stomach and duodenum, and the upper urinary tract are well portrayed.

The technical procedures, the development of the apparatus, the theoretical considerations in the various types of kymography are discussed in great detail. Methods for transforming the shadows shown on the film, made with the kymograph, into more obvious curves are clearly demonstrated. The theory and practice of kymoscopy are also presented in detail. Of considerable interest is the miniature grid which comes with the book. By superimposing this upon many of the outline drawings of kymograms or even upon some of the reproductions of the films the effect of a kymoscope may be obtained. In this way the true nature of the kymogram may be demonstrated; that is, the reversal of the process by which the film was produced will demonstrate the normal movement of the organ examined.

As might be expected, the heart and great vessels receive the major attention. The normal movements are recorded in great detail with thorough discussion of the physiology thus revealed. Considerable experimental work with the kymograph on both animals and man has been done to illuminate many of the problems concerned with the normal and pathologic physiology of the heart. Abnormalities are dealt with most completely.

Among the most interesting chapters is that dealing with the peristaltic waves of the stomach which can be strikingly portrayed by this method.

Altogether, this book is a mine of information on a subject of great importance in the future of roentgenology. To anyone wishing to become familiar with the kymographic method, the volume is invaluable.

WHO'S WHO IN INDUSTRIAL MEDICINE AND TRAUMATIC SURGERY. A. D. CLOUD, Editor. A volume of 289 pages; bound in half leather. Published by Cloud Publishing Company, Chicago, 1936. Price, $9.00.

Industrial medicine has become a specialty in medical practice because of the need and recognition of physicians who will make a study of the conditions around the relations of patients to their employment in industrial life. The study of how best to handle the health and well-being of employees in industry and how to correlate the risks, injuries, diseases, and treatment of same to the best advantage of employer and employee are the *raison d'être* for industrial medicine.

The editor of this book wanted to dignify the field, by letting it be known that there are some, at least, of the more important medical men engaged directly in this field. Among these he recognized many who have done outstanding preventative and curative work. He wanted to publish a book that would, by its own appearance and form, put an end to the abortive directories, where every name in them were paid for by the men named. In this book, *not one of the men whose names are listed, paid a cent for such publication.* It is not that kind of a book. He wanted to make available, for ready reference in libraries, offices, editorial sanctums, etc., information relative to the principal men who are writing on medical subjects, with particular reference to industrial medicine, traumatic surgery, occupational diseases, and allied subjects. He wanted to give a collection of biographical data in the field of industrial medicine and traumatic surgery, which would be in every respect comparable to "Who's Who in America" and evidently he has succeeded in doing exactly that.

The names are arranged alphabetically and geographically (in a supplementary list).

The format, paper, printing, etc., are good. The book will prove to be valuable in many places.

RÖNTGENDIAGNOSTIK DER KNOCHEN- UND GLENK KRANKHEITEN. PART III.—JOINT OSTEOMATOSIS AND CHONDROMATOSIS. By VON PROF. DR. ROBERT KIENBOCK. A volume of 228 pages, 194 illustrations. Published by Urban & Schwarzenberg, Vienna, 1934. Price, RM. 22.50.

The author presents us with a review of the joint osteomas and chondromatosis. It is prepared in the form of an atlas of radiology with a review of the pertinent facts regarding the diagnosis of these conditions. The author presents a complete review of the important literature on each of the phases of the subject and numerous case histories of each type with excellent roentgenographic pictures.

The work is divided into three parts: Part I, joint osteomatosis (which he designates as a "new disease of joints"); Part II, joint chondromatosis (benign), and Part III, malignant chondromatosis and chondrosarcomatosis of joints.

The latter two subjects are ably presented, excellently illustrated and, the writer thinks, cover the present knowledge of this subject in a most excellent manner. Chondrosarcoma, though rare of the joint capsule, rightly has a place in the discussion Reviewing the cases presented in the text, we are led to believe that some of these are primary to chondrosarcomas of the bone rather than of the joint capsule with secondary extension into the joint. The differentiation may be and probably is of academic importance and interest.

As for the "new disease of joints," "joint osteomatosis," we fail to be impressed with the importance or *rationale* of such a nomenclature for it simply represents a grouping together of what we have usually called hypertrophic changes typical of the so-called hypertrophic arthritis or osteoarthritis and of trau-

matic arthritis, and a second group of what he calls solitary and atypical changes which are benign osteochondromas involving or extending into the joints. We cannot see where such a classification helps our knowledge of these conditions but, on the other hand, would say that it seems to us to be confusing.

With it, the author presents an excellent group of cases and a complete review of these case histories, giving us therein an atlas of radiographic reviews of these cases which is valuable to anyone seeking information on this subject.

STRAHLUNGEN: WESEN, ERZEUGUNG UND MECHANISMUS DER BIOLOGISCHEN WIRKUNG. By DR. KARL G. ZIMMER, Assistant an der Strahlenabteilung des Cecilienhauses Berlin-Charlottenburg. A volume of 72 pages, 40 illustrations. Published by Georg Thieme, Leipzig, 1937. Price, 3.20 RM., pasteboard.

In this booklet which is one of a series, "Problems der Theoretischen und Angewandten Genetik und deren Grenzgebiete" (W. F. Reinig, Editor), Zimmer discusses the whole range of radiations fundamentally, completely, briefly, and yet simply. It is an excellent introduction to radiophysics and it gives to all an understanding of the fundamentals of radiation biology.

ABSTRACTS OF CURRENT LITERATURE

CONTENTS BY SUBJECT

Fistulas ... 379
Fractures ... 379
Gall Bladder (Normal and Pathologic) ... 379
Gallstones ... 380
Gas Gangrene ... 380
Gastro-intestinal Tract (Diagnosis) ... 380
General Body Exposure ... 381
Genito-urinary Tract (Diagnosis) ... 381
Grenz Rays ... 381
Gynecology and Obstetrics ... 381
The Hip Joint ... 382
Hodgkin's Disease ... 382
Infections (Therapy) ... 384
Inflammatory Diseases ... 384
The Intestines ... 384
The Lungs ... 385
Osteomyelitis ... 385
Peptic Ulcer ... 386
Postural Defects ... 386
The Prostate ... 387
Radiology, Practice of ... 387
Radium ... 388
Roentgen-ray Burns and Injuries ... 388
Silicosis ... 388
The Skin ... 389
Soft Tissue Roentgenography ... 389
The Stomach ... 389
The Teeth ... 389
The Tonsils ... 389
Tularemia ... 390
Tumors (Diagnosis) ... 390

THE FOLLOWING ABSTRACTORS HAVE CONTRIBUTED TO THIS ISSUE

S. M. Atkins, M.D., of Waterbury, Conn.
G. E. Burch, M.D., of New Orleans, La.
J. E. Habbe, M.D., of Milwaukee, Wisc.
H. A. Jarre, M.D., of Detroit, Mich.
Ernst A. Pohle, M.D., Ph.D., of Madison, Wisc.
William R. Stecher, M.D., of Easton, Penna.
Charles G. Sutherland, M.B. (Tor.), of Rochester, Minn.

CONTENTS OF ABSTRACTS IN THIS ISSUE LISTED ALPHABETICALLY BY AUTHORS

Anderes, E. Roentgenologic Aid in Obstetrics .. 382
Baumann, Robert, and Blond, Kasper. Etiology and Therapy of So-termed Fistula Ani ... 379
Belot, J. General Body Exposure with Roentgen Rays ... 381
Berger, R. A., *with* Hodges, Fred M., jt. auth. .. 384
Best, R. Russell, and Hicken, N. Frederick. Cholangiographic Demonstration of Biliary Dyssynergia and Other Obstructive Lesions of the Gall Bladder and Bile Ducts ... 379
Blaine, Edward S., *with* Glaser, Mark Albert, jt. auth. ... 379
Blond, Kasper, *with* Baumann, Robert, jt. auth ... 379
Butsch, Winfield L., *with* McGowan, John M., jt. auth ... 380
Collins, E. N., and Root, J. C. Elimination of Confusing Gas Shadows during Cholecystography by the Use of Pitressin ... 379
Copeman, W. S. C. Fibrositis ... 384
Dean, H. Trendley. Chronic Endemic Dental Fluorosis (Mottled Enamel) ... 389
Desjardins, Arthur U., Habein, Harold C., and Watkins, Charles W. Unusual Complications of Lymphoblastoma and Their Radiation Treatment ... 383
Dowell, D. Arnold, *with* Kelly, James F., jt. auth ... 380
Dreessen, Waldemar C., and Jones, R. S. Anthracosilicosis ... 388
Dubowyi, E. D., and Gordijan, N. M. Changes in Bacterial Flora of the Tonsils Induced by Roentgen Therapy of Cases Afflicted with Chronic Tonsilitis ... 389
Ettinger, Alice. Visualization of Minute Gallstones: Layer Formation of Bile ... 380
Eusterman, George B. Diagnostic Aspects of Roentgenologically Negative Gastric Disorders ... 389
Gaenslen, F. J. Fracture of the Neck of the Femur ... 379
Glaser, Mark Albert, and Blaine, Edward S. Duration of Fractures and Operative Defects of the Skull as Revealed by Roentgenograms (A Follow-up Study of 100 Patients) ... 379
Gordijan, N. M., *with* Dubowyi, E. D., jt. auth ... 389
Gratz, Charles Murray. Air Injection of the Fascial Spaces: A New Method of Soft Tissue Roentgenography ... 389
Greenbaum, Sigmund S., and Tumen, Henry. Severe Xerostomia from X-ray Treatment for Hypertrichosis ... 389
Grimm, O. The Roentgenological Visualization of the Male Urethra ... 381
Guthrie, Donald, and Sharer, Robert F. Permanence of Cure Following Ruptured Duodenal Ulcer ... 386
Habein, Harold C., *with* Desjardins, Arthur U., jt. auth ... 383

HAMPTON, AUBREY O., and KING, DONALD S. The Middle Lobe of the Right Lung: Its Roentgen Appearance in Health and Disease. 385
HICKEN, N. FREDERICK, *with* BEST, R. RUSSELL, jt. auth. 379
HILDRETH, ROSCOE C., *with* JACOX, HAROLD W., jt. auth. 383
HILDRETH, ROSCOE C., *with* PEIRCE, CARLETON B., jt. auth. 382
HOBART, MARCUS H., and MILLER, DONALD S. Osteomyelitis at Cook County Hospital, with an Appraisal of Orr's Method of Treatment. 385
HODGES, FRED M., and BERGER, R. A. Roentgen Therapy of Some Infections. 384
HUMPHRIS, F. HOWARD. Mild Radium Therapy. 388
JACOBY, GEORG. Grenz and X-rays, and a New Universal Therapy Apparatus. 381
JACOX, HAROLD W., PEIRCE, CARLETON B., *and* HILDRETH, ROSCOE C. Roentgenologic Considerations of Lymphoblastoma: II.—Roentgen Therapy of Hodgkin's Disease. 383
JACOX, HAROLD W., *with* PEIRCE, CARLETON B., jt. auth. 382
JONES, R. R., *with* DREESSEN, WALDEMAR C., jt. auth. 388
JORDAN, SARA M. A Review of the Gastric Ulcer Problem. 386
JOVIN, I. Curie Therapy of Uterine Fibroid and Hemorrhagic Metropathies. 381
KELLY, JAMES F., and DOWELL, D. ARNOLD. Present Status of the X-rays as an Aid in the Treatment of Gas Gangrene. 380
KING, DONALD S., *with* HAMPTON, AUBREY O., jt. auth. 385
KONZELMANN, F. W. Some Discrepancies and Pitfalls that Occur in Clinical Work as the Result of the Grading of Tumors. 390
KORNBLUM, KARL, and TUMEN, HENRY J. What the Radiologist should Know about Clinical Pathology. 387
KRUCHEN, C. The Problem of General Body Exposure to Roentgen Rays. 381
LAPLACE, LOUIS B., and NICHOLSON, JESSE T. Physiologic Effects of the Correction of Faulty Posture. 386
LODGE, W. O. Treatment of Intrasellar Tumors by Radon. 388
McGOVERN, F. H. Primary Tularemic Ulcers in Pharynx. 390
McGOWAN, JOHN M., BUTSCH, WINFIELD L., and WALTERS, WALTMAN. Pressure in the Common Bile Duct of Man: Its Relation to Pain Following Cholecystectomy. 380
McKEEVER, FRANCIS M., *with* WILSON, JOHN C., jt. auth. 386
MAYO, CHARLES W., and WAKEFIELD, E. G. Disseminated Polyposis of the Colon: A New Surgical Treatment in Selected Cases. 380
MILLER, DONALD S., *with* HOBART, MARCUS H., jt. auth. 385
MITCHELL, JOSEPH I. Fracture of the Neck of the Femur in Children. 382
MYERSON, ABRAHAM, and RITVO, MAX. Benzedrine Sulfate and its Value in Spasm of the Gastro-intestinal Tract. 381
NICHOLSON, JESSE T., *with* LAPLACE, LOUIS B., jt. auth. 386
PALUGYAY, J., Roentgen Therapy of Prostatitis. 387
PEIRCE, CARLETON B., JACOX, HAROLD W., and HILDRETH, ROSCOE C. Roentgenologic Consideration of the Lymphoblastoma: I.—Roentgen Pulmonary Pathology of the Hodgkin's Type. 382
PEIRCE, CARLETON B., *with* JACOX, HAROLD W., jt. auth. 383
PENDERGRASS, EUGENE P. The Small Intestine. 384
PFAHLER, GEORGE E. Erysipelas Carcinomatosum Resembling Radiodermatis. 388
RITVO, MAX, *with* MYERSON, ABRAHAM, jt. auth. 381
ROBERTSON, ROBERT CRAWFORD. Acute Hematogenous Osteomyelitis: An Analysis of Seventy-five Cases. 386
ROOT, J. C., *with* COLLINS, E. N., jt. auth. 379
SHARER, ROBERT F., *with* GUTHRIE, DONALD, jt. auth. 386
TUMEN, HENRY, *with* GREENBAUM, SIGMUND S., jt. auth. 389
TUMEN, HENRY J., *with* KORNBLUM, KARL, jt. auth. 387
WAKEFIELD, E. G., *with* MAYO, CHARLES, W., jt. auth. 380
WALTERS, WALTMAN, *with* McGOWAN, JOHN M., jt. auth. 380
WATKINS, CHARLES W., *with* DESJARDINS, ARTHUR U., jt. auth. 383
WEIS, F. H. Concerning the Symptomatology of Congenital Cystic Pulmonary Disease (Honey-combed Lung). 385
WILSON, JOHN C., and McKEEVER, FRANCIS M. Bone Growth Disturbance Following Hematogenous Acute Osteomyelitis. 386

FISTULAS

Etiology and Therapy of So-termed Fistula Ani. Robert Baumann and Kasper Blond. Schweiz. med. Wchnschr., April 11, 1936, pp. 358-361.

Through extensive roentgenographic study of injected fistulæ *in ano*, the authors are convinced that anal fistula should be considered as *thrombophlebitis purulenta chronica ani et recti*. Almost without exception, the typical fistula commences as a peri-proctal abscess, which becomes thrombosed and later causes infected varices. It can be dogmatically stated that in a case in which a fistula is present, hemorrhoids are present. After rupture of the fistula externally, gas and feces escape thereby. The important consideration is the fact that the lining of the fistula is composed of endothelium, partly from the venous walls, and therefore granulation tissue cannot be formed.

WILLIAM R. STECHER, M.D.

FRACTURES

Fracture of the Neck of the Femur. F. J. Gaenslen. Jour. Am. Med. Assn., July 11, 1936, **107,** 105–114.

From the story of the clinical course of the impacted fracture one can best understand the underlying principles involved in the healing process.

No one method of reduction will fit all cases; all cases properly reduced and properly spiked will not go on to solid union. Internal fixation has decided advantages over external fixation and present-day conventional methods, while representing a distinct advance as compared with earlier methods, will give way to more precise and more certain procedures.

The author attempts to rationalize, first, the method of reduction of fractures of the neck of the femur by traction in flexion, and secondly, the method of internal fixation. This attempt is based in part on clinical observations and in part on experimental studies on the skeleton and on animals.

CHARLES G. SUTHERLAND, M.B. (Tor.).

See also under "The Hip Joint," page 382.

Duration of Fractures and Operative Defects of the Skull as Revealed by Roentgenograms (A Follow-up Study of 100 Patients). Mark Albert Glaser and Edward S. Blaine. Jour. Am. Med. Assn., July 4, 1936, **107,** 21–24.

Because of academic and also medico-legal interest, 100 cases were studied by repeated roentgenographic examinations with all technical factors standardized as far as possible. In all children studied, linear fractures disappeared within six months from the time of the injury; in adults, the time varied to as long as eight years. Depressed fractures show obliteration of the fracture lines in from one to four years. In depressed fractures in which fragments of bone are removed the filling defects did not decrease in size. Loose fragments of bone were gradually absorbed. Operative defects acted similarly; the bone never fills in and the edges become smooth and rounded. Osteoplastic bone flaps of the skull turned down for tumor or hemorrhage usually do not degenerate. They are united to the skull by heavy bands of fibrous tissue.

CHARLES G. SUTHERLAND, M.B. (Tor.).

GALL BLADDER (NORMAL AND PATHOLOGIC)

Cholangiographic Demonstration of Biliary Dyssynergia and Other Obstructive Lesions of the Gall Bladder and Bile Ducts. R. Russell Best and N. Frederick Hicken. Jour. Am. Med. Assn., Nov. 14, 1936, **107,** 1615–1619.

Dyssynergia is defined as a spasm of the lower end of the common duct. This and obstructive lesions of the extra-hepatic bile ducts have been demonstrated during operation by cholangiography, *i.e.*, injecting radiopaque fluids into the common duct. This procedure they termed "immediate cholangiography"; the injection of radiopaque substances post-operatively, through catheters, tubes, or fistulas, has been designated "delayed cholangiography." Lipoidine, thorium dioxide sol, and hippuran were found most suitable; each had its advantages and disadvantages. Immediate cholangiograms proved a definite aid in diagnosis and prevented complicating circumstances which arose from incision and exploration of the common duct. The status of the cystic duct can also be ascertained.

Delayed cholangiograms are an aid in determining the status of the choledochal sphincter, the presence of overlooked stones, stricture and tumor, and the presence of pancreatitis or tumor of the pancreas, and also help determine when sufficient time has elapsed for biliary drainage.

CHARLES G. SUTHERLAND, M.B. (Tor.).

Elimination of Confusing Gas Shadows during Cholecystography by the Use of Pitressin. E. N. Collins and J. C. Root. Jour. Am. Med. Assn., July 4, 1936, **107,** 32.

In the course of 600 cholecystographic examinations, the use of this preparation was deemed advisable in 73 instances. They have found the use of pitressin more effective than the use of enemas. In the absence of cardiovascular contra-indications, the dose is one ampoule (10 pressor units). The preparation has not been used in patients having a systolic blood pressure below 100 mm. of mercury or in patients who have advanced hypertension or suspected coronary disease. Cholecystograms were made from 45 to 60 minutes after the injection of the drug. In practically all instances in which the drug was used, intestinal activity was evidenced by mild, cramp-like sensations in the abdomen, together with a feeling of expected defecation, if this did not actually occur.

In those instances in which the blood pressure was taken every five minutes for one hour, there was a gradual and progressive fall in both the systolic and diastolic blood pressure during the first thirty or forty minutes, and then a progressive rise in both until the end of the hour, but in no instance was the terminal reading as high as that before the administration of pitressin.

CHARLES G. SUTHERLAND, M.B. (Tor.).

Pressure in the Common Bile Duct of Man: Its Relation to Pain Following Cholecystectomy. John M. McGowan, Winfield L. Butsch, and Waltman Walters. Jour. Am. Med. Assn., June 27, 1936, **106,** 2227–2230.

Roentgenography was used in the study of the function of the sphincteric mechanism at the lower end of the common bile duct. Direct measurement of changes in physiologic functions of the common bile duct of human beings who had disease of the biliary tract were made by the use of an especially devised manometer. Pressure necessary to force the solution into the duodenum was designated the "perfusion" pressure. When the tube to the reservoir bottle was clamped off, the pressure as recorded in the manometer fell at a variable speed to a fixed level, which was referred to as the "intraductal" pressure. Choledochograms (roentgenograms of the common duct filled with 5 c.c. of lipoiodine) were made before and after the subcutaneous injection of one-sixth grain (0.01 gm.) of morphine to record the findings of the manometric studies.

It was found that administration of morphine produced a rise in pressure in the common duct of patients whose gall bladders had been removed. This rise in pressure was accompanied by pain in one case. Periodic rises in pressure, with pain, were demonstrated independent of the effect of morphine. Amyl nitrite and glyceryl trinitrate relieve the pain and pressure produced by morphine.

CHARLES G. SUTHERLAND, M.B. (Tor.).

GALLSTONES

Visualization of Minute Gallstones: Layer Formation of Bile. Alice Ettinger. Am. Jour. Roentgenol. and Rad. Ther., May, 1936, **35,** 656–661.

Examination of the gall bladder in the upright position and with compression is of value especially in those cases in which the clinical and roentgenologic findings do not agree. This method may reveal small calculi which by gravity will collect at the lowest pole of the bladder; if they are of lesser specific gravity, they may float in a horizontal line above the bile of greater specific gravity, which, in the usual prone position, may not have been seen. This method has also revealed that there is a layer formation in normal bile, and whereas in the prone position much non-contrast bile may cause a faint shadow, in the upright position a normal filling is seen. The loss of non-contrast bile may thus cause an increased density of the bladder after a fatty meal.

S. M. ATKINS, M.D.

GAS GANGRENE

Present Status of the X-rays as an Aid in the Treatment of Gas Gangrene. James F. Kelly and D. Arnold Dowell. Jour. Am. Med. Assn., Oct. 3, 1936, **107,** 1114–1117.

Emergency treatment of a case, using a mobile x-ray unit, with what seemed a small dose when a depth factor was desired, achieved such a brilliant result that eight cases have been similarly treated with startling results. The treatment was successful in cases in which the extremities were concerned; not so successful in cases in which the trunk was involved.

A second series of 32 cases treated with x-rays was reviewed. Eight cases with trunk involvement in this series all lived.

The treatment should be started as soon as the disease is suspected and should be given throughout its course, twice each day for at least three days, morning and evening, and of sufficient voltage to insure penetration of the involved tissue. For an extremity they used from 90 to 100 kv. with 1 mm. aluminum filter, and for the trunk from 130 to 160 kv. with increased filtration; about 100 roentgens per treatment over each area. Serum should also be used unless there is some contra-indication to its use.

CHARLES G. SUTHERLAND, M.B. (Tor.).

GASTRO-INTESTINAL TRACT (DIAGNOSIS)

Disseminated Polyposis of the Colon: A New Surgical Treatment in Selected Cases. Charles W. Mayo and E. G. Wakefield. Jour. Am. Med. Assn., Aug. 1, 1936, **107,** 342–347.

"Polyp" is a gross anatomic term and is not intended to indicate the probable histologic structure of the tumor. Between the extremes of small excrescences on the mucous surface to pediculate tumors of varying size there are many variations in the gross appearance of these tumors. For the sake of clearness the division of Erdmann and Morris into the (1) congenital or adolescent in origin and (2) those which are acquired, or of Wesson and Bargen (1), post-inflammatory and (2) true, is explained. The polyps of the colon that Erdmann and Morris classified as congenital or adolescent in origin are recognized by Wesson and Bargen as true polyps, their cases belonging to this group.

A polyp of the intestine may prove serious in one of four ways: (1) it may bleed as a result of infection or ulceration; (2) it may obstruct the intestine when it has attained sufficient size; (3) it may produce an intussusception, and (4), most important of all, it may become malignant, if carcinoma is not already present when the polyp is first discovered.

Nineteen cases, confirmed by examination after surgical removal, are reported. The surgical treatment by a five-stage procedure has proved the most satisfactory method in the authors' experience.

CHARLES G. SUTHERLAND, M.B. (Tor.).

Benzedrine Sulfate and its Value in Spasm of the Gastro-intestinal Tract. Abraham Myerson and Max Ritvo. Jour. Am. Med. Assn., July 4, 1936, **107,** 24–26.

Benzedrine is a synthetic derivative of ephedrine and in the form of benzedrine sulfate it is stable and can be administered orally. It causes stimulation of the sympathetic nervous system and has been used successfully in the treatment of narcolepsy. In non-toxic doses it lessens or abolishes spastic manifestations of the gastro-intestinal tract within a few minutes. Relaxation of the sphincter permits the passage of a broad stream of the barium sulfate mixture through the pylorus and duodenum. The drug lessens the tone moderately, and diminishes peristaltic activity only slightly. The stomach actually empties more rapidly than normally under the influence of the drug. The lumen of the small and the large bowel is increased, the haustrations become broader and shallower, and contractures or irregularities due to spasm diminish markedly or disappear entirely.

The dosage for the average patient is 30 mg. orally; very stout patients may require 40 mg.; thin and very young individuals are given from 10 to 20 mg. It is given in tablet form with a few sips of water.

Unpleasant effects may occur in about 2 per cent of the patients.

The effect begins in about ten minutes and reaches its height in from fifteen to thirty minutes; it lessens within an hour and gradually disappears.

CHARLES G. SUTHERLAND, M.B. (Tor.).

GENERAL BODY EXPOSURE

The Problem of General Body Exposure to Roentgen Rays. C. Kruchen. Strahlentherapie, 1936, **57,** 54.

A recent interest in the use of general body exposure to roentgen rays induced the author to analyze our present knowledge on this subject. He finds that there are very few data available regarding the mechanism of effect of, and the tolerance for, general body exposure to roentgen rays. There is no question but that the dosage can be regulated very accurately. The regeneration of the blood seems to be satisfactory, provided there does not exist a complicating anemia or cachexia. In leukemia and Hodgkin's disease as well as in polycythemia, this technic seems to be of value. It has also been tried successfully in bronchial asthma. How much can be expected in generalized carcinoma is difficult to predict at this time. General body exposure to roentgen rays should not be given to patients with severe anemia, cachexia, and diseases of the heart or kidneys.

ERNST A. POHLE, M.D., Ph.D.

General Body Exposure with Roentgen Rays. J. Belot. Strahlentherapie, 1936, **56,** 560.

The author discusses the use of general body exposure with roentgen rays in the treatment of skin lesions and malignant disease. Potentials of from 100 to 120 kv. are recommended in dermatologic conditions and from 200 to 300 kv. at 3.5 meters distance for deep therapy. This permits of an even distribution over an area of 1.5 square meters. No improvement in the homogeneity of radiation can be obtained by increasing the filter thickness beyond 0.5 mm. Cu. Although small doses as measured in air are applied, the doses effective biologically are high because of the large surface which is exposed and because of the increase in depth dose due to the long distance.

ERNST A. POHLE, M.D., Ph.D.

GENITO-URINARY TRACT (DIAGNOSIS)

The Roentgenological Visualization of the Male Urethra. O. Grimm. Deutsche med. Wchnschr., 1936, **62,** 1671.

The author relates his experience with the radiological visualization of the male urethra, using "Jodipin" as a contrast medium. The technic of the injection is described in detail. Six roentgenograms are appended showing some of the most characteristic pathology.

ERNST A. POHLE, M.D., Ph.D.

GRENZ RAYS

Grenz and X-rays, and a New Universal Therapy Apparatus. Georg Jacoby. Schweiz. med. Wchnschr., May 9, 1936, pp. 464, 465.

Employing a new apparatus capable of producing rays of a quality between Grenz and x-rays, the author claims to be able to produce results equal to each of these, with the additional convenience of a single apparatus. If the dermatologist wishes to employ either Grenz or x-rays, the machine can readily produce these, so that all types of skin disease can be successfully treated with but one machine.

WILLIAM R. STECHER, M.D.

GYNECOLOGY AND OBSTETRICS

Curie Therapy of Uterine Fibroid and Hemorrhagic Metropathies. I. Jovin. Strahlentherapie, 1936, **57,** 36.

The author reports his experience in the treatment of 47 cases of uterine fibroids and bleeding in 47 cases. The dose for fibroids varied from 1,400 to 4,200 mgh., depending somewhat on the size and location of the tumor. Gamma rays were used (1 mm. Pt). The patients with uterine bleeding were subdivided in four groups: hemorrhage during puberty, in the child-bear-

ing period, climacteric bleeding, and bleeding after the menopause. In the first group, doses of 480–720 mgh. seemed sufficient; in the second group, doses of 1,000–1,680 mgh. were necessary; in the third group this was increased to 2,400–2,880 mgh. In the last group the doses amounted to 4,500 mgh. in order to have sufficient radiation in cases in which an intra-uterine malignancy might be present.

In conclusion, the author states that radium and roentgen rays are equals from the therapeutic standpoint. Roentgen rays have the advantage of no mortality and no morbidity. Radium therapy is indicated in cases with excessive bleeding, in carcinoma of the cervix complicated with fibroid, in submucous fibroids if operation is refused, in uterine bleeding in virgins, occasionally during the child-bearing period, and in uterine bleeding during the menopause.

ERNST A. POHLE, M.D., Ph.D.

Roentgenologic Aid in Obstetrics. E. Anderes. Schweiz. med. Wchnschr., Jan. 11, 1936, pp. 26-28.

The author emphasizes the discrepancy between the fetal position *in utero* as depicted in text-books and as it is constantly noted roentgenographically. The acute flexion of the torso, hitherto considered the position of the fetus, had been proven to be erroneous. It was probably resultant of study of the prepared specimen, in which fixation has caused uterine musculature contraction and pressure upon the fetus. In all cases noted roentgenographically there was the appearance of relaxation of the fetus, thereby permitting rotation of the body until the last day of gestation. If this motion is interfered with, due to diminished or abnormal amniotic fluid, anomalies such as club-foot, etc., are prone to result. Additionally, roentgenography has shown that with rupture of the amniotic sac, the fetus assumes an extended position of the vertebral column thereby aiding fetal axis pressure during delivery.

A diagnosis of pregnancy can be made under most unusual and favorable circumstances as early as the eighth or ninth week, but absolute proof is not present until the eighteenth week. This has become of less importance since the advent of the Aschheim-Zondek test.

The most practical aspect of radiology in obstetrics is pelvimetry. Of the numerous complicated methods advocated, the author considers only two to be of simple practical worth, namely, the anteroposterior projection of Martius, with the patient in the semi-sitting position and the superior pelvic strait parallel to the film. This is accomplished by making the distance from the top of the symphysis pubis to the table top, and from the spinous process to the table top, equal. A check upon this is readily accomplished by noting, in the roentgenogram, whether the ischiac spines are situated midway between the symphysis pubis and the promontory of the sacrum. An additional profile roentgenogram, as advocated by Guthmann, completes the pelvimetric measurements. The sagittal plane of the conjugata vera is made parallel to the film by adjusting the patient so that the central ray is directed through both trochanters. This is also a check upon the finished roentgenographic position, by noting whether or not there is superimposition of the trochanters. Since all these measurements are magnified and distorted, they are readily corrected by employing a simple geometric relationship between the distance of the conjugata vera to the distance of the superior strait and the target from the film. Although theoretically it is as important to measure the fetal head by cephalometry, the author considers this procedure as very unsatisfactory.

Roentgenograms are useful in detecting various pelvic malformations. Although one can deduce with fair accuracy the age of the fetus by noting the ossification centers present, this is often a difficult procedure due to superimposition of the fetal outline upon the maternal skeleton. Multiple pregnancies and fetal monstrosities are diagnosed with certainty, and hydrocephalus, microcephalus, and anencephaly are worthy of especial attention. If the latter is demonstrated roentgenographically, the mother can be spared the chagrin and arduous labor of giving birth to a monster by the production of a therapeutic abortion. Prior to Cæsarean operations a roentgenogram should always be obtained to determine whether or not there is intra-uterine fetal death, a condition which is readily diagnosed by noting overriding of the cranial bones of the fetal head, out of proportion to the estimated time of gestation, with marked extension of the fetal spine.

WILLIAM R. STECHER, M.D.

THE HIP JOINT

Fracture of the Neck of the Femur in Children. Joseph I. Mitchell. Jour. Am. Med. Assn., Nov. 14, 1936, **107**, 1603–1606.

Fractures of the neck of the femur are relatively infrequent in children. These may be divided anatomically into three types: (1) epiphyseal separation, (2) transcervical fracture, and (3) cervico-trochanteric fracture. Traumatic separation of the upper femoral epiphysis is rare; the cervico-trochanteric type is the most common. The fracture may be incomplete or the fragments may be impacted by the violence of the force. Bony union occurs promptly, as a rule, but often with coxa vara deformity, which causes subsequent disability of a serious nature. Treatment by continuous traction, combined with immobilization of the hip in abduction, is advised to prevent coxa vara and shortening of the extremity.

CHARLES G. SUTHERLAND, M.B. (Tor.).

See also under "Fractures," page 379.

HODGKIN'S DISEASE

Roentgenologic Considerations of Lymphoblastoma: I.—Roentgen Pulmonary Pathology of the Hodgkin's Type. Carleton B. Peirce, Harold W. Jacox, and

Roscoe C. Hildreth. Am. Jour. Roentgenol. and Rad. Ther., August, 1936, **36**, 145–164.

An analysis of all cases of primary diseases of the hematopoietic system other than anemia occurring at the University of Michigan Hospital between the dates of July 1, 1925, and Feb. 1, 1935, was made by the writers. During this interval of approximately ten years, 681 cases have been examined. While there was a slight increase in number per year there was no relative increase when compared with the number of patients registered. There were 214 cases classified as either (1) simple Hodgkin's type, of which there were 172, (2) Hodgkin's disease with leukemia cutis, of which there were 12, (3) Hodgkin's disease with acute leukemia, of which there was one, and (4) sarcomatous Hodgkin's disease, of which there were 29. X-ray classification of cases based on chest radiography was as follows: (*a*) no chest examination, 16 cases; (*b*) normal chest roentgenograms, 74 cases; (*c*) hilar adenopathy, 22 cases; (*d*) mediastinal tumor, 46 cases; (*e*) mediastinal tumor and hilar adenopathy, 8 cases; (*f*) parenchymatous involvement, 6 cases; (*g*) parenchymatous involvement and hilar adenopathy, 10 cases; (*h*) parenchymatous involvement and mediastinal tumor, 8 cases; (*i*) parenchymatous, hilar, and mediastinal involvement, 3 cases; (*j*) pleural involvement with or without either of the above, 22 cases. There are numerous illustrations which emphasize the necessity of lateral as well as postero-anterior radiographic examinations. Quite commonly roentgenographic evidence of intrathoracic involvement is far greater than the clinical signs or symptoms of the chest disease would indicate.

Since the x-ray manifestations of intrathoracic Hodgkin are so variable, it follows that positive differential diagnosis of Hodgkin from other commoner intrathoracic diseases cannot be made roentgenologically. Similarly, the x-ray findings offer no basis for prognosis as to duration of the disease.

Approximately one-third of the cases in this series had normal chest x-ray findings.

J. E. Habbe, M.D.

Roentgenologic Considerations of Lymphoblastoma: II.—Roentgen Therapy of Hodgkin's Disease. Harold W. Jacox, Carleton B. Peirce, and Roscoe C. Hildreth. Am. Jour. Roentgenol. and Rad. Ther., August, 1936, **36**, 165–168.

In a series of 215 cases of Hodgkin's disease treated during the past decade, Group I (58 patients) received repeated local irradiation, Group II (50) were irradiated over the entire lymphatic system, Group III (53) had only one or two local (obviously inadequate) treatments, and Group IV (54) received no treatment in this hospital, by the authors. About the same number of each of these four groups had received some radiation before being admitted to the University of Michigan Hospital. In the first group, local radiation was applied repeatedly to the areas involved, with either medium or short wave length roentgen rays with dosage from 100 to 400 r per portal, at intervals of approximately one month. In Group II, general systemic radiation including all lymph-gland-bearing areas was employed, with medium wave length x-rays to the peripheral gland structures and short wave length x-rays to the deeper glands of the chest, abdomen, and pelvis. The dosage was from 200 to 500 r per field depending upon the patient's age and general clinical condition, and 12 to 15 portals, at the rate of two per day, constituted each course. This series was repeated after approximately six weeks, unless the patient had made exceptional improvement from the original series.

The irradiation of the patients in Group III was obviously incomplete, and the cases placed in Group IV were utilized as controls.

From the analysis of this series of cases the authors conclude that roentgen therapy in any degree has induced definite extension of life, as compared with untreated patients, and furthermore, that systemic radiation has accomplished a better symptomatic response than local radiation but is no more effectual than local treatments in prolongation of the total duration of the disease.

J. E. Habbe, M.D.

Unusual Complications of Lymphoblastoma and Their Radiation Treatment. Arthur U. Desjardins, Harold C. Habein, and Charles W. Watkins. Am. Jour. Roentgenol. and Rad. Ther., August, 1936, **36**, 169–179.

Hodgkin's disease with primary involvement of the abdominal nodes is much more common than generally is realized. Furthermore, when there is infiltration into structures in cases in which lymphoid tissue does not occur normally, many other diseases may be simulated. The writers consider chronic infection (commonly pyogenic, less commonly tuberculous or syphilitic) to be the probable etiologic factor, and point out that the first gland enlargement is usually in that part of the body where the infection was harbored.

Complications personally observed include fever, pruritus, infiltrations of the skin, orbital infiltrations, sternal erosions by mediastinal gland enlargements, recurrent laryngeal nerve involvement, pleural effusion, ascites, edema of lower extremities, infiltration of testis, bone involvement either by direct erosion or invasion, infiltration of the spinal cord or brain, and secondary infection associated with the lymphoblastoma.

Fever and pruritus usually indicate involvement of the retroperitoneal nodes, and these symptoms respond promptly with thorough radiation of the abdominal glands. In abdominal therapy, four fields anteriorly and four fields posteriorly should be employed with the rays converging toward the mid-line where most of the glands are situated. Medium voltage rays (135 kv.p.) using 4 or 6 mm. aluminum filter are preferred because of the greater safety of repeated series than when 200 kv.p. therapy is utilized from the beginning.

The more acute the process the less satisfactory the response to irradiation. In addition to chronicity,

thoroughness of the treatment is the next most important factor in influencing the duration of the disease. Maximal doses are to be avoided, however.

In cases in which the rather rare complication of secondary infection with pus formation has taken place it is important to surgically drain the pus; otherwise radiation will be ineffective.

J. E. HABBE, M.D.

INFECTIONS (THERAPY)

Roentgen Therapy of Some Infections. Fred M. Hodges and R. A. Berger. Jour. Am. Med. Assn., Nov. 7, 1936, **107,** 1551–1554.

This is a report of accomplishments and conclusions obtained through 17 years of experience. To offer the patient the utmost that irradiation will give there must be complete and absolute co-operation between the co-workers.

Various lesions were collocated in accordance with their sensitivity or susceptibility to irradiation as follows: Group 1, Early localized erysipelas in adults; furuncles and furunculosis; granulomas; infected hemangiomas; cellulitis of certain types; Mikulicz's disease; parotitis, and rhinophyma. Group 2 embraced carbuncles, blastomycosis, and sporotrichosis.

Group 1 comprises those infections which are sufficiently amenable to irradiation to render no other form of therapeusis necessary. The lesions respond so readily that one may predict an immediate amelioration or abortment, followed by rapid regression and healing.

In Group 2 they have placed those infections in which irradiation is an important auxiliary in the armamentarium for the management of these lesions.

Different types of lesion and similar lesions in different stages of development react somewhat differently to roentgen rays. The technic employed is not empirical; every case is individualized and treated accordingly.

CHARLES G. SUTHERLAND, M.B. (Tor.).

INFLAMMATORY DISEASES

Fibrositis. W. S. C. Copeman. Jour. Am. Med. Assn., Oct. 17, 1936, **107,** 1295–1297.

Fibrositis is the term given to an inflammatory reaction of fibrous supporting tissue to extraneous poisons either toxic or bacterial. It is characterized by nodular or bandlike thickenings in the affected fibrous tissue, in the fascia lining the muscles or joints, or sometimes embedded in the substance of the muscles themselves. In the latter case, they can be felt only when the muscles are in a state of relaxation after the passing of the acute attack.

Fibrositis may attack fibrous tissue wherever it is situated, and since such tissue may occur almost anywhere in the body the manifestations of this disease are protean.

Roughly, the situations in which it occurs with frequency may be thus classified: (1) Subcutaneous tissue (panniculitis) which will include the areolar and adipose tissue found in this situation. (2) Intramuscular, which will include inflammation of the tissue that separates the fascial muscular planes (myositis), the lining of the bursæ (bursitis), and the periarticular fibrous tissue (capsulitis). (3) The fibrous sheaths of the chief nerve trunks, such as the sciatic or the brachial nerves (interstitial neuritis).

Fibrositis is most common in sites in which a combination of strain and chill occur most usually. Common situations are, therefore, the lumbar region (lumbago), the back of the neck, the shoulder, the scalp (rheumatic headaches) and the elbow (certain forms of tennis elbow). Less common sites which when affected often give rise to mistaken diagnoses are the attachment of the iliocostalis muscle to the lower six ribs, and the subcutaneous tissue of the precordial area.

Biopsy reveals no typical pathologic structure. Whatever the actual etiology of fibrosis may be, the background will nearly always be found to reveal chronic fatigue or strain.

Clinically there appears to be two stages in most cases of fibrositis: (1) The stage of effusion; in cases in which this is superficial, a puffy swelling may be seen; (2) the stage of organization, during which the serofibrinous exudate gets partially absorbed, while the residue becomes invaded by fibroblasts, and a low grade fibrosis results, forming intramuscular and interfascial adhesions and, later, palpable nodules. A further frequent complaint is of muscular stiffness, which generally persists even after the pain has disappeared.

Lewellyn, in the majority of his cases, confirmed the presence of either enlarged or shrunken thyroid glands.

Treatment includes a salt-free diet, increase of fluid intake, heat, and massage.

CHARLES G. SUTHERLAND, M.B. (Tor.).

THE INTESTINES

The Small Intestine. Eugene P. Pendergrass. Jour. Am. Med. Assn., Dec. 5, 1936, **107,** 1859–1861.

It is essential that collective studies be made of the small intestine in healthy individuals and in patients having lesions of the small intestine. Lesions involving other portions of the gastro-intestinal tract, as well as conditions outside of it, may exert a profound influence on the mechanics and pattern of the small intestine. Any investigation, therefore, should include a consideration of all such factors. The author presents a plea for a standard meal, so that one radiologist may compare his results with those of another. The pattern of the small intestine will vary considerably, depending on the composition of the foodstuff it receives. It varies also with the consistency of the meal, its size, the gastric emptying time, and the tonicity of the intestinal tract. Certain pathologic conditions, which only indirectly affect the intestinal tract, may cause a profound change in the small intestine pattern.

The author recommends a meal consisting of 5 ounces (140 gm.) of barium sulphate and 5 ounces (150 c.c.) of water. The normal stomach empties such a meal in the majority of instances within two hours; occasionally the period is as long as three hours. The head of the column reaches the cecum occasionally in one and one-half hours. In most instances it requires from two to three hours to reach the cecum and from four to six hours for the intestine entirely to empty itself.

The mucosal pattern is described in detail in the different portions of the small bowel. The upper three-fifths of the small intestine consists of jejunem, and this generally lies to a large extent in the left side of the abdomen. The lumen varies in size in fat and thin individuals and in men and women.

The examination of the small intestine requires several hours as a rule, and occasionally its physiologic function is disturbed as a result of the patient having developed a headache because of food being withheld. Psychic or emotional disturbances may apparently influence the motility and pattern of the small intestine. Normal variations and anomalies were found more frequently than anticipated.

The radiologist should be extremely careful in attempting to identify for the surgeon the point of obstruction in the small intestine.

Various drugs were found to have a profound effect on the gastro-intestinal tract for from twenty-four hours to five days.

A large group of other lesions that disturb the normal physiology reflexly are being studied, to be reported later.

CHARLES G. SUTHERLAND, M.B. (Tor.).

THE LUNGS

The Middle Lobe of the Right Lung: Its Roentgen Appearance in Health and Disease. Aubrey O. Hampton and Donald S. King. Am. Jour. Roentgenol. and Rad. Ther., June, 1936, **35**, 721–739.

The writers describe the roentgenologic findings in 56 patients suffering from disease of the right middle lobe, 41 of whom were operated on or had postmortem examination, while 15 had bronchoscopy or lipiodol bronchography only. Particular consideration is devoted to the appearances of various pathologic processes as shown on the right lateral film.

First attention is given to the applied roentgen anatomy, and it is pointed out that in cases in which the lesion has more or less the distribution of the lateral aspect of the lobe a triangular shadow will be produced on the lateral roentgenogram, but if the lesion follows the distribution of the medial part of the lobe a more rectangular shadow will result. A lesion of the latter distribution will obscure more or less the right heart border and render cloudy the right cardiophrenic angle.

Primary encapsulated interlobar empyema is now considered a rarity by the writers as in the entire series not a single proven case was found. Lung abscess in the same region is fairly common by comparison. A warning is offered against the careless misinterpretation of interlobar empyema when actually there is no lesion in this vicinity but merely an overlap of the right diaphragm and posterior aspect of the heart in such a way as to give a shadow simulating effusion. This appearance is most readily obtained if for any reason the right diaphragm is abnormally elevated.

Not infrequently, the right middle lobe is so fibrosed and collapsed as to be of no more than two to three centimeters in greatest thickness in lateral projection; in such cases interlobar pathology is apt to be diagnosed mistakenly. The demonstration of the interlobar pleural septa is of great importance in making for correct differential diagnosis, although at times bronchoscopy or bronchography is absolutely essential to a final diagnosis.

J. E. HABBE, M.D.

Concerning the Symptomatology of Congenital Cystic Pulmonary Disease (Honey-combed Lung). F. H. Weis. Fortschr. a. d. Geb. d. Röntgenstrahlen, September, 1936, **54**, 230–237.

This is a report of two cases of this relatively rare disease with considerable references to the literature (Zdansky, Lenk, Canigiani, etc.). One of these patients was under observation for over nine years and in spite of repeated intercurrent infection ultimately showed no progress in numerous pulmonary cysts. Roentgen therapy, collapse therapy, and paralysis of the diaphragm were without influence. The other patient, representing the type of small rounded and polyhedral cysts throughout both lungs, succumbed to intercurrent infection (tuberculosis) and could be studied carefully at autopsy. This infection involved only the right middle lobe. It is assumed that in this case a chronic peribronchitis led to progressive interstitial induration with subsequent alveolar ectasia. The necessity of etiologic diagnosis of pulmonary cavities is apparent.

H. A. JARRE, M.D.

OSTEOMYELITIS

Osteomyelitis at Cook County Hospital, with an Appraisal of Orr's Method of Treatment. Marcus H. Hobart and Donald S. Miller. Jour. Am. Med. Assn., Oct. 3, 1936, **107**, 1118–1121.

The authors present an appraisal of Orr's method of treatment in this institution. More than 375 patients were operated on and of these only one-third have answered the follow-up requests to return for a final check-up. The cases reviewed number 108, with 135 admittances to the hospital; the majority occurred in adults between the ages of 20 and 40. The Orr method gave 62 per cent symptom-free results in these cases.

CHARLES G. SUTHERLAND, M.B. (Tor.).

Acute Hematogenous Osteomyelitis: An Analysis of 75 Cases. Robert Crawford Robertson. Jour. Am. Med. Assn., Oct. 10, 1936, **107,** 1193–1199.

The essential pathologic condition in acute hematogenous osteomyelitis is a blood stream infection complicated by local infection of one or more bones. The author presents a review of 75 successive, unselected, private, and clinical cases treated within the past nine years. Definitely acute, hematogenous, pyogenic cases only were included. Twenty-two days was arbitrarily chosen as the time limit in determining acuity. The principle of early drainage was followed in all of these. Early roentgen changes were seen as early as the fifth day and were absent as late as the fourteenth day following onset in one case.

The majority of the best results were obtained by drainage of the bone within one week following the onset. The mortality rate was also highest in cases in which drainage was instituted within this period.

CHARLES G. SUTHERLAND, M.B. (Tor.).

Bone Growth Disturbance Following Hematogenous Acute Osteomyelitis. John C. Wilson and Francis M. McKeever. Jour. Am. Med. Assn., Oct. 10, 1936, **107,** 1188–1193.

This is a review of 64 patients with 90 individual foci of infection, who suffered from acute hematogenous osteomyelitis. Fifty-nine in this group showed involvement of the long bones with a diaphysis and one or more terminal epiphyses. Five cases showed the lesion in the os calcis, which grows by accretion and has no diaphysis or terminal epiphyses.

The growth disturbances occurring in the long bones were classified as follows: (1) Primary variations: (*a*) perimetric hypertrophy; (*b*) lengthening; (*c*) shortening; (*d*) directional changes (coxa valga and anteroposterior and lateral bowing). (2) Secondary variations; disturbance of joint inclination (genu valgum, medial deviation of the ankle or lateral deviation of the ankle). (3) Concomitant variations: compensatory lengthening of uninfected paralleling bones and shortening and diminution of size of feet without infection in any of the component osseous structures.

Early operative intervention in this series did not decrease the frequency of growth disturbances.

CHARLES G. SUTHERLAND, M.B. (Tor.).

PEPTIC ULCER

A Review of the Gastric Ulcer Problem. Sara M. Jordan. Jour. Am. Med. Assn., Oct. 31, 1936, **107,** 1451–1456.

The author reviewed 119 cases in which the diagnosis of gastric ulcer was made on a reasonable basis and checked by surgical exploration in 31 cases, or by follow-up studies varying from a few months to eleven and one-half years after medical treatment.

After the data of history and chemical changes were analyzed and their adjuvant value estimated, the x-ray examinations assume the decisive rôle in differentiation. The important x-ray criteria are of two kinds: (1) the original appearance of the lesion by x-rays and (2) changes in the original appearance of the lesion with trial treatment. It is undoubtedly true that the lesion of the pars media which appears in the original x-ray examination as the protruding crater off the lesser curvature in the pars media and without involvement of the posterior wall is practically always benign and shows a tendency to relatively quick healing, with synchronous diminution of the size of the crater. Complete healing occurs when there is complete disappearance of the crater.

With all the diagnostic data (history, chemistry, and x-ray examinations) at hand, one is still not infrequently doubtful as to the benignity of the lesion. Safeguards are a limited period of trial management and a careful follow-up of all cases.

CHARLES G. SUTHERLAND, M.B. (Tor.).

Permanence of Cure Following Ruptured Duodenal Ulcer. Donald Guthrie and Robert F. Sharer. Jour. Am. Med. Assn., Sept. 26, 1936, **107,** 1018–1022.

The authors discuss the opinion of various schools as to the best procedure in acute perforation of a duodenal ulcer. They review their own series of 78 cases, four operated on outside the hospital and 74 treated in the hospital, over a 26-year period.

Pneumoperitoneum, determined by correct fluoroscopic examination, was of distinct value in many cases, and Singer and Vaughan are quoted as maintaining that it is effective in 86 per cent of their cases proved by operation. The treatment employed in their cases was one of simple closure, a gastro-enterostomy being performed at a later date only if obstruction developed. Four patients, however, required a gastro-enterostomy at the first operation because of obvious obstruction of the duodenum. Their operative mortality was 20.8 per cent.

CHARLES G. SUTHERLAND, M.B. (Tor.).

POSTURAL DEFECTS

Physiologic Effects of the Correction of Faulty Posture. Louis B. Laplace and Jesse T. Nicholson. Jour. Am. Med. Assn., Sept. 26, 1936, **107,** 1009–1012.

Observations were made on a series of 26 healthy adults of both sexes having disorders of the back attributable to postural strain.

Twenty-three subjects were studied with respect to the immediate physiologic effects of changing from a faulty posture to a corrected posture; eight of these were further studied with respect to the degree of permanence of these effects following a year of routine corrective exercises.

It was concluded that the results of correcting faulty posture differ widely between individuals, irrespective

of the grade of the defect. A correct posture appears to be an appreciable advantage to circulatory and respiratory function in the majority of persons, but in some a postural defect may be a compensatory mechanism which it is inadvisable to disturb. The therapeutic application of postural correction should be made according to the requirements of the individual case, and only after an attempt to determine in what posture the individual is functionally most efficient.

CHARLES G. SUTHERLAND, M.B. (Tor.).

THE PROSTATE

Roentgen Therapy of Prostatitis. J. Palugyay. Fortschr. a. d. Geb. d. Röntgenstrahlen, September, 1936, **54**, 215–218.

This report is based on a study of 76 cases of which eight were afflicted with acute or subacute prostatitis, 41 with a chronic infection; in 11 cases the infection occurred in a hypertrophic prostate, and 16 cases were referred for radiation therapy under the assumption of a malignancy in the gland. Seven of the acute cases harbored an urethroginous infection, while in the eighth case it followed a severe cellulitis of the hand. Six of the 41 chronic cases harbored a gonorrheal infection, some of them a mixed infection; in three cases a so-called aseptic prostatitis had to be assumed.

Six of the eight acute cases were cured by a single series of radiation. After an unsuccessful first series, the two other patients did not return for further observation or treatment. Of the 41 chronic cases, 15 were cured by one series, though a second series was given to six of them to secure complete disappearance of subjective distress; eight further cases were cured subjectively and objectively by two series; three additional cases by three series of radiation. In two cases an abscess had to be drained after the first series of radiation. Four cases developed a recurrence after the first series and required a second series of treatment several months later. In six cases no effect of any kind was obtained by the first series of radiation, and this method of treatment was then discontinued. In three cases there was an aggravation of symptoms rather than an improvement.

Thus, a cure was obtained by from one to three series of radiation in more than 75 per cent of acute and chronic cases; 22 per cent of the cases showed no effects. In three cases an aggravation occurred.

Technic.—Small doses, highly filtered irradiation, large field of entry, total dosage—the smaller, the more intense the inflammatory reaction. On admittance, from 20 to 60 r from a large suprasymphyseal field, and equal amounts to a sacral field from six to eight days later, to which may be added, if necessary, a smaller perineal field following an additional interval of from three to six days. The series may be repeated after three weeks, at which time the total dose may be increased somewhat.

Quite frequently a considerable irradiation-reaction is observed even after such small doses. Under the assumption of prostatic malignancy 16 cases were referred, but on the basis of the resulting reaction and cure, the diagnosis had to be corrected to that of inflammatory prostatitis.

From his observations the author comes to the conclusion that prostatitis is one of the most gratifying conditions for well directed roentgen therapy, in the acute as well as chronic state. In dubious cases of malignancy, radiation therapy would seem justifiable for therapeutic and diagnostic purposes.

H. A. JARRE, M.D.

RADIOLOGY, PRACTICE OF

What the Radiologist Should Know about Clinical Pathology. Karl Kornblum and Henry J. Tumen. Am. Jour. Roentgenol. and Rad. Ther., August, 1936, **36**, 202–232.

In his effort to become master of the art of roentgenology the radiologist is apt to forget much of the general medical knowledge which he learned in undergraduate days or before limiting his work. Furthermore, as the years pass there is much that is new in all the specialties and it is difficult for the radiologist to keep abreast of all these advances. Clinical pathology is one of the most important specialties concerning which he should keep informed. It is with the idea of fulfilling in part this need that the writers present this material with a page and a half summary at the end, in table form, of a number of ranges of normal in laboratory tests of the blood, the stomach contents, and the liver, gall-bladder, and kidney functions.

Concerning blood examinations, the reporting of hemoglobin determinations in grams per 100 c.c. is advocated in preference to percentage.

Since most malignant tumors are accompanied by an increased sedimentation rate, this test is advocated as a means of aiding in the differential diagnosis of tumors.

In cases of anemia in which there is a pathologic condition of the bone marrow irradiation should be given with caution. However, there is no substantial proof to date that the diagnostic use of thorotrast will cause injury to the blood or bone marrow.

The writers also prefer to see differential counts of the white blood cells in actual number per centimeter rather than in percentages.

In considering blood chemistry, it is pointed out that the most common extra-renal cause of an increase of urea nitrogen is acute high intestinal obstruction.

If liver disease can be excluded, the serum phosphatase determination may be of special value in studying bone diseases. For example, in Paget's the phosphatase is markedly elevated. The same is true of hyperparathyroidism. In myeloma there is lesser elevation.

In Schüller-Christian syndrome, a condition dependent upon a hyperlipemia, there is usually also a hypercholesteremia.

The presence of alkalosis may aid materially in final evaluation of obstructive lesions at the pylorus found

roentgenologically. If there is an alkalosis associated with an obstructive lesion at the gastric outlet, the lesion is probably benign, since in most cases of benign ulcer there is a hyperacidity and the vomiting will cause a loss of chlorides. Conversely in malignancies with achlorhydria there is no chloride loss and no alkalosis.

Gastric analysis curves are important in duodenal ulcer in which lesion from 70 to 80 per cent of the cases there is a sharp rise in free HCl which remains elevated in the first hour and may go even higher in the second and third hours, and in gastric carcinoma, in from 60 to 80 per cent of which cases there is an achlorhydria.

Concerning liver tests, it is emphasized that there is no functional test, which if negative, will entirely rule out advanced hepatic disease.

Gall-bladder drainage and cholecystography are not antagonistic but if used jointly will often supplement one another in diagnostic information. If by gall-bladder drainage one can demonstrate cholesterol crystals and the orange pigment of what is probably calcium bilirubinate, one may be nearly 100 per cent certain of the presence of gallstones. The absence of concentrated B-bile in repeated drainages constitutes good evidence of disturbed gall-bladder function.

J. E. HABBE, M.D.

RADIUM

Treatment of Intrasellar Tumors by Radon. W. O. Lodge. British Med. Jour., Dec. 19, 1936, pp. 1257, 1258.

The author describes his experience and operative technic with radon seed implantation in treating intrasellar tumors. A case is described in which the patient was well two years after the operation.

G. E. BURCH, M.D.

Mild Radium Therapy. F. Howard Humphris. British Med. Jour., Sept. 12, pp. 532–535.

Various methods are discussed for collecting and using radium emanation in practice. The author considers it beneficial in (1) "gout and goutiness"; (2) certain gynecological conditions, as pelvic pain and congestion, salpingitis, and in the treatment and cure of sterility; (3) certain dermatological conditions, as psoriasis, mycosis, fungoids, eczema, and lichen; (4) chronic catarrhal conditions and affections of the sinuses and respiratory organs, and (5) for "revitalizing" in convalescence, notably from septic troubles. "It is eminently suitable in the complaints of middle and old age, premature aging more especially in recuperation, in cases of exhaustion or weakness following operation, long illness, or overwork."

He discusses the various theories by which radon gas is said to produce its beneficial results. He concludes: "These findings suggest, as a possible *rationale* for emanotherapy, that the emanation, by virtue of its positive and negative charges, may remove the electric charges from the body colloids, and have a stabilizing action resulting in a sedative effect on the tissues. The *rationale* of the radon treatment of rheumatism is one of oxidation and detoxication . . . and there is no doubt but that treatment by mild radium therapy does raise the patient's resistance in a remarkable way."

G. E. BURCH, M.D.

ROENTGEN-RAY BURNS AND INJURIES

Erysipelas Carcinomatosum Resembling Radiodermatitis. George E. Pfahler. Am. Jour. Roentgenol. and Rad. Ther., June, 1936, **35,** 804–812.

Since in most cases, at least, this form of malignancy develops independent of x-ray therapy or other local application, it is reasonably certain that irradiation has nothing to do with the condition. Nevertheless, it is sometimes misdiagnosed as radiodermatitis or radiotelangiectasis. While in some cases the disease extends through the blood vessels of the skin by direct permeation, the more common route is through the lymphatics of the skin and subcutaneous tissue. Microscopic study of biopsy material shows collections of cancer cells in the corium with no invasion of the epidermis. The clinical appearance is that of malignant disease with an inflammatory reaction. Roentgen irradiation may give definite improvement, although in 15 cases examined and treated by the author none was permanently cured.

J. E. HABBE, M.D.

SILICOSIS

Anthracosilicosis. Waldemar C. Dreessen and R. R. Jones. Jour. Am. Med. Assn., Oct. 10, 1936, **107,** 1179–1185.

This is a study based on practically all the personnel of three representative mines engaged in mining and processing of hard coal in Pennsylvania, numbering 2,711 tuberculosis-free, disabled former anthracite workers in hospitals intensively investigated, and limited observations on the disease as observed in a tuberculosis sanatoriums in Pennsylvania. The last occupation listed was the miner's present job. The weighted average dust exposure in each job was multiplied by the number of years in that occupation. The sum of all these products divided by years in the industry, gave a fairly accurate estimate of the man's average exposure. The workers were divided chiefly into three groups: (1) Regular miners exposed to a dust with a free silica content of from 3 to 4 per cent. (2) Workers in the haulage ways, exposed to dust with a free silica content of 13 per cent. (3) Rock tunnelers and muckers, exposed to 35 per cent free silica.

Of this group of 2,711, they found 616 (22.7 per cent) affected with anthracosilicosis. The prevalence of clini-

cal pulmonary tuberculosis in those with anthracosilicosis was 15 per cent in the early cases and 43 per cent in the late, well established cases. Among the controls and those essentially negative for anthracosilicosis, the prevalence was, respectively, 1 and 2 per cent, or about the same as in the general population.

CHARLES G. SUTHERLAND, M.B. (Tor.).

THE SKIN

Severe Xerostomia from X-ray Treatment for Hypertrichosis. Sigmund S. Greenbaum and Henry Tumen. Jour. Am. Med. Assn., Oct. 17, 1936, **107,** 1297.

In various parts of the United States a number of patients with untoward cutaneous complications from the use of the commercialized Tricho (x-ray) System in the treatment of hypertrichosis of the face have been observed. Filtered x-rays obviated the superficial complications such as cutaneous atrophy and telangiectases so commonly seen. It was, however, followed by deep effects on the salivary glands of the oral mucous membrane and their secretions.

The authors report a case of a woman treated by a "cosmetologist" with progressive development of oral dryness.

CHARLES G. SUTHERLAND, M.B. (Tor.).

SOFT TISSUE ROENTGENOGRAPHY

Air Injection of the Fascial Spaces: A New Method of Soft Tissue Roentgenography. Preliminary Report. Charles Murray Gratz. Am. Jour. Roentgenol. and Rad. Ther., June, 1936, **35,** 750, 751.

Air injections into the fascial spaces are described by the writer in this preliminary report. Like the encephalogram, the "pneumofasciagram" may have both diagnostic and therapeutic value. Fascial adhesions may produce certain restriction of motion and will cause certain abnormal distribution and irregularity of the air shadow. The freeing of adhesions by this diagnostic procedure may account for the therapeutic benefit that the writer has seen in some cases. The procedure should take place in the operating room, and the patient should remain hospitalized from 24 to 48 hours.

J. E. HABBE, M.D.

THE STOMACH

Diagnostic Aspects of Roentgenologically Negative Gastric Disorders. George B. Eusterman. Jour. Am. Med. Assn., Oct. 31, 1936, **107,** 1432–1436.

The efficiency of modern roentgenologic diagnosis permits classification of diseases of the stomach into roentgenologically positive and roentgenologically negative disorders. The former (ulcer, cancer, and so on) constitute about a fifth of the cases of chronic dyspepsia coming under observation at the clinic; roentgenologically negative disorders which have been classified and described constitute the remainder.

Gastric disturbances reflexly engendered by disease of the abdominal viscera other than the stomach itself or its continuations exceed in importance the gastric neuroses, because of their nature and extent and the comparative frequency of their occurrence. They constitute from a third to two-fifths of all cases. The neuroses constitute about a fourth of the total.

CHARLES G. SUTHERLAND, M.B. (Tor.).

THE TEETH

Chronic Endemic Dental Fluorosis (Mottled Enamel). H. Trendley Dean. Jour. Am. Med. Assn., Oct. 17, 1936, **107,** 1269–1273.

The endemic hypoplasia of the permanent teeth known as chronic endemic dental fluorosis, or mottled enamel, is a water-borne disease associated with the ingestion of toxic amounts of fluorides in the water used for cooking and drinking during the period of calcification of the affected teeth. The permanent teeth in particular are affected, although in areas of medium to marked severity the signs of mottled enamel are at times observable on certain of the deciduous teeth.

Mottled enamel is a permanent physical disfigurement. The distribution is world-wide. The most severely affected State in the United States is Texas. Among the foreign countries the Argentine Republic is the most seriously affected.

In spite of their defective structure, mottled enamel teeth exhibit no greater liability to caries than do normally calcified teeth.

In the light of present knowledge, this disease is readily preventable. The logical approach to the solution of this problem is avoiding the use of water containing fluorides in excess of the permissible limit. In some instances, this can be accomplished by simply changing to a readily available source in the same neighborhood that is free from toxic amounts of fluoride. The water may be diluted to bring the fluoride content down, or the water supply may be treated by chemical means for the removal of toxic amounts of fluorides.

CHARLES G. SUTHERLAND, M.B. (Tor.).

THE TONSILS

Changes in the Bacterial Flora of the Tonsils Induced by Roentgen Therapy of Cases Afflicted with Chronic Tonsillitis. E. D. Dubowyi and N. M. Gordijan. Fortschr. a. d. Geb. d. Röntgenstrahlen, January, 1936, **53,** 73–76.

The bacterial flora of 79 patients afflicted with chronic tonsillitis was investigated before and after roentgen irradiation, up to four months after treatment. The following results were reported:

Group 1.—This group consisted of 54 cases afflicted with chronic tonsillitis but without disease of the internal organs or joints. Before roentgen therapy, 27 cases showed the presence of hemolytic streptococcus; in 19 cases (70.4 per cent) this infection disappeared after the first treatment, while *Streptococcus viridans* disappeared in seven of twelve cases, and *Staphylococcus aureus* in six of nine cases.

Group 2.—This group consisted of 11 patients with chronic tonsillitis and disease of the internal organs, or bones and joints: *Streptococcus hæmolyticus* disappeared in five of eight cases, *Streptococcus viridans* in three of five, and *Staphylococcus aureus* in six of nine.

Group 3.—This group consisted of 14 children with high-grade tonsillar hyperplasia and respiratory difficulties but without angina. No pathogenic microorganisms were present in the tonsils, either before or after roentgen irradiation.

Treatment employed consisted of 210 kv., 4 ma., 0.5 mm. of copper plus 2 mm. of aluminum, 25 cm. focal-skin distance, size of field 4 × 5 cm. in children; 5 × 6 cm. in adults, over the mandibular angle. Children from 5 to 10 years of age received 25 per cent of a skin erythema dose; 10 to 15 years of age, 30 per cent; older ones, 33 to 35 per cent. One skin erythema dose equaled 640 r. A second series of treatments was given usually one month after the first.

H. A. JARRE, M.D.

TULAREMIA

Primary Tularemic Ulcers in Pharynx. F. H. McGovern. Jour. Am. Med. Assn., Nov. 14, 1936, **107,** 1629, 1630.

Tularemia is primarily a fatal disease of rodents, chiefly rabbits, and is secondarily transmitted to man from rodents by the bite of an infected tick and other blood-sucking insects, by contamination of the hands with infected material, or by the ingestion of improperly cooked infected meat. It occurs in four main types: ulceroglandular, oculoglandular, glandular, and typhoidal. The ulcers are chronic indurations resembling the primary lesion of syphilis, appearing after an incubation period of from one to nine days, and accompanied by chills, high fever, sweating, headache and prostration. The regional adenopathy, often quite large and frequently suppurative, may appear before the primary lesion is apparent. Pulmonary manifestations consisting of pulmonic consolidation, bronchitis, or pleural effusion may follow the initial symptoms. A moderate leukocytosis with a distinct shift of the Schilling count to the left is found. A diagnosis is ordinarily confirmed by a positive agglutination reaction of the serum against *Bacterium tularense.* This reaction becomes positive generally in the second or third week and persists indefinitely.

One case is reported with primary ulcerations in the pharynx and bilateral cervical adenopathy, the inoculation presumably occurring from crushing an infected dog tick and carrying the organisms to the mouth on the fingers.

CHARLES G. SUTHERLAND, M.B. (Tor.).

TUMORS (DIAGNOSIS)

Some Discrepancies and Pitfalls that Occur in Clinical Work as the Result of the Grading of Tumors. F. W. Konzelmann. Am. Jour. Roentgenol. and Rad. Ther., June, 1936, **35,** 795–803.

Reasons for discrepancies between histologic grading of degree of malignancy by the Broders' method and the end-result of treatment are discussed. A single biopsy may be misleading as to the predominating cell type; hence the tumor grade. Moreover, Broders' original work was done on material which did not have the advantage of modern x-ray and radium therapy. Again, the biopsy will give no clue as to whether metastasis has occurred or not, a fact of obvious vital importance in arriving at a prognosis.

In lip cancer, while in a general way size of the lesion is comparable with duration of tumor and degree of malignancy, the coincidence is not sufficiently frequent to permit grading on any one factor.

According to some workers, changes in grade may occur in metastasis. In the opinion of others, histologic grading has been unsatisfactory both as a means of estimating prognosis and for determining radiation dosage.

Even in the hands of a group of expert tumor pathologists histologic grading resulted in an accuracy of only about 50 per cent as compared with the clinical follow-ups in a series of 100 breast tumors.

There are, of course, many well known exceptions to Bergonié's law which states that the more anaplastic tumors are, the more radiosensitive they are. The situation is made worse by misapplication of the terms "radiosensitivity" and "radiocurability."

J. E. HABBE, M.D.